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TINNITUS AURIUM, ETIOLOGY, DIFFERENTIAL DIAGNOSIS, TREATMENT AND REVIEW OF TWENTY-FIVE CASES.

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The subject of tinnitus aurium is not only a problem for the otologist, but also demands the interest of every medical and surgical specialty. In this paper I shall endeavor to delve into the most important available literature related to the study of this topic. It is my object to combine into one comprehensive survey many points of view of the etiology and treatment of noises in the ears associated with or without deafness. I have found in my study of this significant problem that, as a general rule, only one point of view has been given by a good number of authors. The writer feels that a complete general analysis of the most related and important facts ought to be made. This, I feel, will aid those who wish to make further studies in this field. More constructive research and study must be pursued along the questions of tinnitus and deafness if patients are to respect the specialty of otology. Many innocent patients are rapidly falling into the clutches of exploiting quacks because these sufferers are not properly relieved.

I would define tinnitus aurium as a subjective sensation of sound in one or both ears, which is a common symptom of aural disease, generally encountered in disturbance of the cochlear mechanism or from numerous other causes which I

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will discuss further in this manuscript. There are two types of tinnitus; the first is external, originating in the middle ear and produced by mechanical factors which create actual sounds as spasmodic contraction of the tensor tympani muscles. The second type is internal tinnitus, which has no associated sound, but at the same time begins in the labyrinth and is created by some upset or metabolic equilibrium of the nerves, or the disease of the related and associated apparatus.

The most practical theory of sound has been presented by Helmholtz,[1] and he explained it as a theory of sympathetic resonance. He noticed that a string of a piano could be made to vibrate when the same note was sung loudly to it, and stated that resonating apparatus of the ear was the basilar membrane. This membrane increases twelve times in breadth as it winds from the base of the cochlea to its tip. The reason the basilar membrane is widest at the tip is that the bony shelf, the lamina spiralis, leaves a gap between its extremity and the bony wall of the cochlea, which is much greater at the apex than at the base. The basilar membrane bridges this gap. The basilar membrane is not a true membrane, but is composed of a large number of tightly stretched transverse strands varying in length according to the size of the gap between the lamina spiralis and the bony wall of the cochlea. The entire basilar membrane is computed approximately to contain about 24,000 such strands, gradually increasing in length from the base to the tip of the cochlea. The sum total of this structure very much can be compared to the strings of a piano. Each strand of the basilar membrane is thought to have its peculiar vibration rate and it is assumed that such vibrations are communicated to a corresponding branch of the cochlear nerve, from which place the impulse is carried to the brain. Helmholtz believed that the basilar membrane is able to analyze each compound wave motion in the perilymph into its constituent simple wave; each of these simple waves sets into vibration that strand of the basilar membrane which possesses exactly the same wave length, acting in accordance with the known laws of sympathetic resonance. It is believed that each strand, vibrating to a simple tone of its own wave length, excites the hair cells which lie on it and, through them, the nerve fibers. The tectorial membrane perhaps acts as a damper, suppressing the vibration after the causative force has ceased to operate. Accordingly it has

been computed experimentally that the range of the human ear comprises from 6000 to 11,000 different tones. The Organ of Corti[2] is more than sufficient to accommodate this range, as there are 24,000 strands in the membrane, and 14,000 nerve fibers communicating with the hair cells. However, the brain does not perceive sound as a phenomenon occurring in the ear, but refers the sound to the external source of the sound waves, which is similar to what takes places in the instance of vision. Experimentally it has been proven that the destruction of the apex of the cochlea causes loss of appreciation of low notes and destruction of the base of the cochlea produces loss of appreciation of high notes. In the range of hearing the highest audible tone corresponds[3] to 30,000 to 40,000 vibrations a second, the lowest to about 30 per second. Between these limits as numerous as 6000 variations of pitch can be perceived.

Apparent pitch of tinnitus is 3500 cycles per second, the actual frequency of discharge of nerve fibers is a matter of 600. The indications are that the apparent pitch of the sound is dependent primarily on the place of the membrane, where it is reached rather than the frequency of the impulse traveling over a single nerve fiber. This does not preclude[4] the possibility of a 3600 cycle component in a large number of staggered 600 cycle impulses sent over a bundle of nerve fibers situated close together. This has been clearly shown by Wever and Bray in their experiments at Princeton.

Babbitt[5] expressed his opinion that the source of tinnitus was thought to be recognized in the acoustic cerebral centers. The complexity of influences forming the syndrome of tinnitus renders it impossible to isolate distinctive pathology. Functional or organic abnormalities appear definitely casual in the following conditions, such as Eustachian tube, tympanic cavity types, etc.

Tinnitus[6] may or may not be associated with deafness. Ringing noises may precede, accompany or follow the onset of deafness; the sounds may be continuous or intermittent, increasing or diminishing in severity. When taking a history, it is significant to inquire whether the symptom complex is associated with deafness, dizziness or staggering.

Noises in the ear may be simple or compound in nature. If it is simple in type, the subjective sound is uniform in

character and still may vary in its intensity. In this classification[7] are placed the blowing, whistling, humming, hissing and rustling sounds. These various noises are worse when the individual is fatigued and are more pronounced when in quiet or noisy places.[9] In some cases the sounds are in the form of a specific tune, as is found in a music teacher suffering from tinnitus. Tinnitus aurium, when it is bilateral, may be found in neurasthenia[10] and in patients afflicted with high blood pressure. In these they complain of the noises being more in the head. Ringing in the ears[11] may be clicking in nature, in which case the tympanic membrane may have rhythmic contractions. Some otologists emphasize the fact that anything which varies the normal endolymph pressure, whether upward or downward, may produce tinnitus. When compound tinnitus is present it is comprised of subjective sounds which vary in character and loudness. In this type the noise is of sudden, violent, rapping, rumbling, shrieking and jangling high pitched effect. Compound sounds exist in sequence of prolonged cases of middle ear deafness and may precede cochlear involvement. The patient sometimes describes these sounds to be so unbearable as to drive him mad. High pitched sounds are often associated with otosclerosis.

Objective sounds are produced by violent contraction of muscles of deglutition on Eustachian tube or by contraction of stapedius or tensor tympanum muscles. This condition is found in neurotic and neurasthenic individuals. When the examining surgeon can himself hear the buzzing, throbbing, clicking noises of constant or intermittent nature, it is termed an objective sound. These are associated with vertigo and seasickness. The objective type of tinnitus may be found in defective circulation, as in an aneurysm of the carotid, which sounds can be stopped temporarily by constriction. In aortic constrictions,[12] since cardio-insufficiency frequently supervenes in these cases, the symptoms are overtaxed and altered circulation when the blood pressure to the head and upper extremities are easier and more free than that found in the lower parts of the body; therefore, violent pulsations, plethora with continual tinnitus headaches and epistaxis are developed. If the tinnitus has a systolic bruit and is synchronous with the heartbeat, it is stopped by carotid compression; it may be related to vasomotor paralysis, aneurysm or inflammation of the middle ear.

The anterior wall of the tympanum is separated from the internal carotid by a thin bony plate only and the tympanic floor is in relation with the jugular fossa which lodges the bulb of the jugular vein. As an abnormal congenital condition the carotid canal may project into and encroach upon the cavity of the tympanum and it is not difficult to conceive that any malposition in this direction might give rise to sounds which the patient could not fail to hear. In the tympanic floor dehiscences may exist either as a congenital defect or as a result of an old necrotic process which has undergone resolution. Many such cases have been observed during surgical operations and in the course of postmortem examinations. Blood sounds are of a low medium pitch; pulsating noises are of arterial origin. Venous sounds are nonpulsating and the sounds are uniform, occuring in long continued anemic states. The sounds are of a low continuous hum which are supposed to be due to an altered condition of the blood in its passage from the sinus into the bulb of the jugular.

Noises vary with atmospheric pressure, humidity and temperature.[13] When a patient is in a dry elevated and warm temperature the noises are least noted, becoming louder on sea level, where it is cold and damp. In some cases tinnitus aurium may be present just after treatment following inflation in a chronic catarrhal deafness when the tightness of Eustachian tube loses its congestion.

Regarding the types of sound and origin, it has been discovered that gurgling, boiling and whistling qualities are associated with diseases of the rhinopharyngeal catarrh with the involvement of the Eustachian tube and middle ear, or may be associated with some sort of irritation in the external meatus. When the sound is constant, rushing, knocking in character, it is said to be due to a congestion or hemorrhage of the labyrinth. Ringing or roaring type of tinnitus probably is caused by chronic middle ear catarrh, tympanic disease, lues, brain tumors or meningitis. When noises[14] are referred to the head (tinnitus cerebri) rather than the ear, and neither deafness nor aural disease is present, it may be caused by either arteriosclerosis or meningitis.

The most common causes of tinnitus are found in states of neurasthenia, neuropathic diathesis or in patients who suffer from vasomotor irregularity; also in such manifestations

as diseases of the ear, as in impacted cerumen in the external auditory canal, acute serous otitis media,[15] obstructive tubotympanitis and in chronic catarrhal otitis media. It is frequently found in diseases due to anemia and leukemia and cerebral anemia, and is also commonly found in toxic states, as in gout, nephritis, alcoholism, delirium tremens, excessive use of tobacco, senile arteriosclerosis and in patients who have disturbances of digestion.

The less frequent causes of tinnitus aurium are in hypertrophy of the heart; aneurism or cerebral or basilar or vertebral arteries, which produces a pulsating tinnitus; in premonitary aura[16] of epileptic seizure, or tinnitus may initiate an attack of catalepsy, or may be found in the onset of hysteria. It is also found in insanity, at which time there are hallucinations of all sorts of noises and whispering sounds. There are also natural stages of hallucinations due to long continued duration of tinnitus, which may disappear suddenly and reappear. Tinnitus[17] is less commonly associated with vertigo and deafness, which is the chief manifestation in Meniere's disease; in an attack of migraine, preceded or accompanied by sudden loud noises; in intracranial hemorrhage or thrombosis there are frequently noises present. Tinnitus is encountered not uncommonly in organic disease involving auditory nucleus[18] or tract, when there may be a tumor pressing over the tract. Cushing[19, 26] vividly demonstrated the importance of tracing chronologically the symptoms in order to distinguish the group of acoustic tumors from other tumors, such as gliomas, meningiomas and other rarer forms which may invade the lateral recess. In typical acoustic tumor conditions, the first symptom is likely to be tinnitus in one ear accompanied or soon followed by increasing deafness on the same side. After an interval, which is perhaps several months (but may be a matter of years), other cranial nerves in the neighborhood may be involved by pressure so that numbness of the face and absent corneal reflex on the affected side are almost the rule. Eventually the facial nerve is apt to be involved as well as the XIIth cranial nerve; in such a case dysphagia and imperfect utterance results.

Tinnitus is not uncommon in meningitis cerebri, especially so in pachymeningitis. Ringing in the ears may be present following sunstroke, as well as in chronic alcoholic patients

and those who at one time received serious injuries to the skull.

Bagley[20] reported an interesting case of a girl who developed a head murmur at the age of 16 years and when she became 20 years old she died and the postmortem revealed considerably dilated venous sinuses, and the branches were found in deep excavations made in the skull.

Not[21] infrequently drugs like quinine, salicylates, ergot, asperin, alcohol, mercury, caffeine, carbon disulphide, arsenic and morphine have a peculiar selective action on vessels of the ear and the eye. Sollmann[22] recorded some cases where a hypodermic inoculation of morphine sulphate gr. ¼ produced a marked tinnitus which disappeared in a short time. Not only has this been experimentally produced on animals, but this has been experienced by many clinicians. In cinchonism[23] the first symptom is tinnitus and deafness, and loss of sight follows within a few hours. The pupils become widely dilated and inactive to light and the retinal arteries are reduced in size. The disc and fundi are pale. Complete loss of consciousness may occur in severe cases of cinchonism.

Tinnitus[24] in delirium tremens comes on gradually with premonition signs and symptoms. There may be a tinnitus in both ears which further develops into various sounds or cries of human voices of scolding or threatening nature.

In[25] osteitis fibrosa or leontiasis ossea, when the face is affected, such symptoms as headache is chief complaint, accompanied by a marked tinnitus, a hammering subjective noise is experienced in the head. Neuralgia, deafness and blindness occur in some conditions. Deformities of the head and face assume different degrees with exophthalmus.

When[26] pressure from a pineal tumor is directed further backward, involving the posterior quadrigeminal regions, such auditory signs as ringing and buzzing in the ears are commonest. Loss of hearing is bilateral, but is unequal in degree. There is associated with this early in the manifestation a stiffness of the neck with some opisthotonos, giddiness, eye palsies, vomiting, retinal changes and chocked disc.

In cerebellopontine[27, 28] angle tumors, which most often originate in the endoneurium of the acoustic nerve, frequently

the patient presents himself with a complicated tinnitus of musical nature together with vertigo as first signs, followed by progressive nerve deafness. The next nerve which is involved is the trigeminous (Cushing). This condition has about 20 per cent mortality on patients who are operated upon in acoustic nerve conditions, which is lower in proportion claimed in any other intercerebellar tumor involvement.

Patients[29] in whom cerebral aneurysm is present have symptoms which are very vague and rarely recognized until the sac has ruptured. The premonitory symptoms appear before the danger sign of paroxysmal headaches that might have been mistaken for migraine; also there are present tinnitus, epistaxis associated with signs of pressure on the optic nerve. Rupture usually occurs due to sudden effort, like running or jumping.

Children[30] rarely have tinnitus, for it has been found that the vestibular aqueduct is scarcely very wide, allowing an uninterrupted flow of endolymph into the saccus endolymphaticus and a wide cochlear aqueduct, permitting free passage into the subarachnoid space. This provides a situation which tends to maintain one even pressure on the endolymph. It is found, however, in the practice of pediatrics that tinnitus is due to acute catarrh of the tube and tympanum, and associated with this is a depression of hearing. Sometimes the noises are unaccompanied by pain or temperature. In[30] such cases as in acute infections, especially scarlet fever, measles, diphtheria, influenza and typhoid fever, where primary disease of internal ear is a complication, a toxic neuritis results and symptoms such as deafness and tinnitus appears from the involvement of the cochlea. There[31] is also a disturbance of the equilibrium, such as vertigo, nystagmus, nausea and involvement of the vestibular apparatus. It has been observed that mumps may upset the cochlear and the vestibular apparatus. Toxins from malarial or typhus fever may cause a tinnitus.

In recent years clinicians found tinnitus not only created but aggravated by foci of infection in the tonsils, sinuses, mastoid, appendix or the teeth. Focal[33] infections of the mastoid[32] antrum wall has caused upsets in the general constitution and local absorption into the auditory nerve. There are linqual tonsillitis and pharyngitis of chronic nature that may

give rise to noises in the ear. 200[34, 35] cultures were taken from infected teeth at the time of extraction and only two contaminations were made, the balance showed 144[34] strept. viridans, 19 S. Fecalis, 23 S. Salivarus and six nonhemolyticus. The author feels that there has been considerable overestimation about the foci of infection idea, but care must be taken to rule it out first, before other causes are sought.

Symptoms[36] resulting from affections of the auditory nerve are tinnitus, deafness, giddiness, vertigo and other signs found associated with the ear, such as otorrhea, pain in the head, facial paralysis and herpes due to the involvement of the geniculate ganglion. The deafness may precede or follow the condition and it may or may not be continuous or intermittent, increasing or decreasing in severity. The deafness may be unilateral or bilateral. In a series of 915[37] cases of toxic neuritis that were studied, 455 were found to have anemia and 271 of these complained of vertigo. Tinnitus due to disturbance in special sense of hearing is often difficult to separate clinically from vertigo, of which it is frequently a symptom (Osler[38]). The two conditions are usually associated, but it is better to distinguish them as an entity by themselves. Tinnitus was found in cases where patients suffered from pernicious anemia.

Bandler[39] quotes Martin, who found symptoms following castration and this produced a rushing of blood to the head, combined with a feeling of anxiety, marked tinnitus aurium, palpitation, dizziness and sweating. These symptoms occur in different combinations and as many as three times per day. After two or three years these symptoms disappear.

Moore[40] presented a case of pregnancy where tinnitus appeared about the seventh month and disappeared soon after she gave birth, with no recurrence of this distressing condition.

At the time of climateric some types of female patients suffer from most annoying tinnitus aurium with hot flashes accompanied with other vasomotor symptoms. This disturbance is associated with deficiency of internal secretion of the ovaries.

Endocrine[41] disturbances associated with conditions such as abnormal menstruation, pregnancy, parturition, muscular[42] exhaustion and fatigue, following walking, talking, sneezing,

chewing, abnormal movements of the head, will aggravate tinnitus. Sometimes after a heavy meal the condition of ringing in the ears will be made worse. Over indulgence of sexual life and menorrhagia will cause marked ringing in the ears also. Out of 585[47] cases studied at Evans Memorial, there were incidence of tinnitus in 36.6 per cent of endocrine patients, and 32.7 per cent in nonendocrine cases. Noises in the ears is not a characteristic symptom in ductless glandular diseases, but is seen equally often in hypofunction of each of the endocrine glands. Thyroid gland upset, especially where deficiency sets in, usually causes a generalized infiltration in which state a cellular growth and oxidation is affected in the entire human body. When the infiltration affects the auditory nerve and vestibular apparatus, tinnitus results without any definite ear lesions. Sajous[48] believes it is caused by anemia of this special sense organ, accounted for by low blood pressure and by a depleted oxidase, which is its oxidizing principal, causing impairment of hearing associated with tinnitus. Adrenal insufficiency may produce anemia and mental fatigue or congestion of the labyrinth.

Tinnitus[43] aurium is encountered in patients suffering from hypertension, in prealbuminuric state of Bright's disease or latent arteriosclerosis, presclerosis or hypertensive cardiovascular disease. All[44] of these terms certainly indicate that various pathologic involvements accompany or are entailed by the increase in blood pressure.

Observations disclose that tinnitus is produced following lightning[45] stroke. Such patients complain of hearing defects too. Occasionally rupture of the tympanic membrane has been not an infrequent sequelae of lightning stroke.

In general[46] malnutrition, hysteric neurasthenic states, tinnitus is often encountered (Spear).

Tinnitus[45] with dizziness presents itself in hookworm disease with associated symptoms, such as irregular menstruation, hemic murmurs, venous pulsations of the neck, with weak and easily compressible pulse.

Roget[42] states that adhesions of Eustachian tube to posterior pharyngeal wall frequently will cause noise in the ears.

Occupational tinnitus is associated with deafness. In noisy occupations, slight deafness is often noticed after the first

exposure, but its effects disappear if the occupation is continued. Early in the disease tinnitus is present but does not last long. After reaching a certain stage of partial deafness, the affliction remains stationary or advances very slowly. Even if the patient soon takes up a quiet occupation, the deafness is permanent. The susceptibility of noise deafness differs much in various individuals. At times there are concurrent vestibular reactions resulting from exposure to noise, as in railway engineers and boiler makers. Loud reports from cannons, explosions have similar effect but usually are of a temporary nature unless repeated frequently. Noises of factories must be regarded as a traumatic agent affecting the cochlear apparatus, and the gravity of the affection depends from the alternation of the work and rest periods; from the condition of the nose and type of nasal breathing. Losanov[49] found that an extra day of rest given during the midweek to rabbits upon which he experimentated helped to decrease the complete destruction of organ of Corti when the experimental work was conducted daily for a period of five months. He also compared the clinical data with experimental ones and he discovered that affections of phonoreception of individual workers employed in noisy manufactories were caused by destruction of organ of Corti with atrophic degeneration of peripheric neuron. It has been found that the perception of combined sounds (low voice and especially the whisper suffers most and can be explained by traumatization of Corti organ). Losanov further stated that from his experimental work and clinical observations, acousticotraumatic processes develop more slowly in men than in rabbits. Industrial workers employed in noisy places have been found to be lacking in higher and lower tones. Mental strain often is associated with a patient who has tinnitus with deafness and he may brood on his troubles which may later lead to insanity.

Hallucinations, commonly defined as perceptions with no actual objective stimulus to account for them, and may exist in sensory organs or sensory tracts. In conditions of otosclerosis the patient complains of vague noises in the ears. Sometimes the noises are described as running water or whistling or ringing of bells and are mostly confused. Usually in psychotic tendencies the patient feels he hears shots fired or thinks voices are talking to him. In shell[50] shocked soldiers, bursting[51] of powerful explosives will leave horrible noises in

the subconscious mind and yet no organic lesions are present. Tinnitus is, in a number of cases, psychic or neurasthenic in origin. Woakes[53] brings forward an argument to prove that Mohammed and Joan of Arc were probably sufferers from tinnitus induced by damp and unhealthful conditions under which they lived. This idea is amusing but helps to lead back to illusions. Parish states that in auditory delusions the lowest degree is represented by the psychic hallucination. These are soundless internal voices which seem to the subject to be addressed to him. These sounds as experienced by the insane are spoken of as spiritual or soul language. No vocal hallucinatory[53] noises, *e.g.*, ringing of door bells, or steps in the hall, or in the room itself, seem, as a rule, to be indistinguishable in intensity from corresponding objective sounds. Sometimes in dreams the hallucinatory noises are to be sufficiently loud as to awaken the sleeper. In such cases, dealing with external noises in the head with abnormal intensity, is a state of dissociation and not hallucinations. Dr. MacLaurin[91] of Australia, in "Mere Mortals," states that Martin Luther was tormented by infernal crashings and whistlings in his ears. Luther awakened from sound sleep and yelled back at his devil. At one time he threw an inkpot at this infernal being. A spot is still seen on the wall of Wartburg. These noises were accompanied by attacks of giddiness which caused him to fall at times and interfered with his work. As he grew older his deafness became worse and with this his cardiac distress. Dr. MacLaurin wonders whether Luther's severe tinnitus was not perhaps the cause of his serious and militant zeal in his religious ideas and belief. It might be that if Martin Luther had not been a victim of Meniere's disease that there, perhaps, might not have been a reformation.

Otosclerosis[55] (capsulitis labyrinthi) may have the associated condition of deafness with or without noises; pain, stuffiness in the head may also be present. It is a disease of unknown etiology, characterized pathologically, by spongification of the bony capsule surrounding the labyrinth, and clinically, by progressive deafness and severe tinnitus. Otosclerosis in a luetic patient runs a much more rapid course and leads to an earlier and much more severe type of deafness. The most frequent site of the lesion is just above and in front of the oval window. Bony changes taking place in this region may result in ossification of the annular ligament

with fixation of the stapes which leads to loss of motion of this ossicle. The important phenomenon in this disease is the paracusis Willisiana, *i.e.*, the patient hears better in noisy places than in quiet ones.

Jones and Knudsen[56] quoted statistics of 340 cases of hearing impairment analyzed with a higher percentage in conductive than perceptive lesions and 59 per cent of these cases had a history of tinnitus aurium.

Relaxed[52] conditions of the temperomandibular parts of the mandible on one or both sides, and impacted teeth, can cause tinnitus aurium.

Peter McBride[54] pointed out that patients' ears are frequently exposed to loud noises, as in the case of boiler makers, artillery men and other occupations where there are exposures to terrific noises. Often, repeated concussions of the labyrinth tends, at least, to cause deafness; not infrequently the labyrinth lesions are associated with middle ear catarrh. The loud sounds act by drawing the drum membrane inward and with it the stapes. Owing to Eustachian tube obstruction, the air within the tympanum is rarified. Thus, the common association of boiler makers' disease of deafness is probably accounted for. In these cases the deafness is generally associated with tinnitus and vertigo. In more than thirty years since McBride wrote, no advancement has been made of any importance concerning the connection between ear-splitting noises and tinnitus accompanying one's regular vocation. Patients complain of high pitched tinnitus much more often than tones of lower scale. It is possible that the center of high pitched sounds being the last acquired evolutionary are therefore more vulnerable.

Certain cases of tinnitus are caused by abdominal tumors, malignant growths, which act reflexly. P. R. Wrigley removed a mass and found tinnitus disappeared. In like manner, persistent forms of noises in the ears may be due to visceral disturbances in other parts of the body. Disorders of pelvic and abdominal cavities are also factors which may create the ringing in the ears. Woakes[53] explains in hypothetical manner the nervous relationship between the viscera and the labyrinth. The stomach and other abdominal viscera are largely supplied by the pneumogastric nerves and

the communication between the vagus and inferior cervical ganglion is established by means of a fasciculus from the vagus to the ganglion in question. Thus, in certain disorders of the stomach, *e.g.*, impulses may be sent by way of the inferior cervical ganglion to the labyrinth whereby its arteries are caused to dilate. Woakes claims that under such conditions the blood supply to the area involved may be ten times as great as under normal conditions.

Tinnitus may follow simple mastoid, as is the case in conditions of the last period of postoperative treatment of simple mastoid operation after the perforation in the tympanic membrane has healed, and some time before the postauricular wound has closed the patient may complain of a very disturbing tinnitus aurium and often deafness (Kopetzky[58]).

Certain types of noises in the ears may arise from the tympanic membrane. It has been observed that there are conditions of progressive deafness in ringing in ears which are due to resistance of sound waves and to altered elasticity characterized by interstitial[59, 60, 61] myringitis. When[62] there has been an elimination of resistance of various parts and segments of tympanic membrane, it results in improvement of the hearing from different frequencies. Retraction of the drum head from tensor spasm does not cause tinnitus because it does not raise the intralabyrinthine pressure as long as the stapedius muscle is intact. Kato proved in his experimental work that a tone stimulus of a definite intensity excites reflex contractions of both muscles irrespective of pitch, though the stapedius is more sensitive and first to respond. When the stapedius muscle atrophies and the joint ankyloses, severe subjective noise may be present. In early conditions these patients may be given relief by otomassage and Faradisation. The tensor and stapedius are stripped skeletal muscles controlled by upper and lower motor neurones, either of which may go out of action with resultant spasmodic or flaccid paralysis.

A case of persistent tinnitus following a fracture of the base of a skull is recorded and Dr. Frazier's operation[63] of severing the auditory nerve distally; following such a condition at first glance would seem to bear out the contention of those who maintain that tinnitus is caused by a lesion within the labyrinth. For four months the patient was compara-

tively free from noises in the head after severing the auditory nerve. The recurrence of the tinnitus shows, without doubt, that in this case there occurred four months later a further regeneration of the auditory nerve central to the part of the sectioning, and thus was capable of producing tinnitus. Before the operation it would be reasonable to believe that the process attacked cells within the spiral ganglion peripheral to the point of sectioning. This would account for the patient's temporary recovery from the tinnitus. Subsequently ear tests proved that the auditory nerve was completely severed and yet tinnitus recurred. This would indicate that tinnitus is, in part, probably produced by a further toxic involvement of ganglionic cells along the course of the auditory nerve tracts, or at their termination in the cerebral cortex. There are other cases recorded where severing of the auditory nerve distally did not relieve the patient.

In a[36] series of 25 cases of tinnitus aurium which I examined and studied, the analysis disclosed as follows: The age varied from 17 years to 73. The greatest incidence was in the age limits between 40 and 78 years. The least between 17 and 35 years. These patients were not employed in any occupations which were of any noisy nature. All the patients were white; five patients were Russians, 12 Americans, two Austrians, two Polish, one Roumanian, two were Italians and one was born in Holland. All gave a history of tinnitus of a variable nature. Some described the noises as being very intense and aggravating. On the other hand, few patients stated that the tinnitus aurium was very mild and almost imperceptible. 15 out of the 25 cases had either one or both ears involved as regards the defective hearing and these were getting progressively worse. Four patients gave a history of hay fever and one gave a history of bronchial asthma. Five complained of frequent colds. Two had a chronic discharge from the ears. One elderly patient gave a history of diabetes over a period of about 15 years. One young patient reported that he was operated on by Dr. Cushing for a meningioma, after which procedure he had a total loss of hearing on the right side, with a marked diminished hearing of the left side, associated with a loud and shrill tinnitus on the latter side. From 14 patients were elicited the history of chronic nasal discharge with constant hawking of thick mucous material from the throat. Five patients reported chronic sore throats

and eight had dental complaints for several years with negligence for dental care. Four individuals were constipated. All denied veneral disease, but when blood Wassermanns were taken it was found that two out of the 25 cases showed four plus Wassermann.

The findings in the above cases following examinations were that 16 of the series had deflected septums. Some were very markedly deflected. Six showed diseased and pyorrhetic teeth with numerous cavities. Two had chronic discharging ears (O.M.P.C.). One had O.M.P.A., and one had a polyp in one ear. 20 cases showed bilateral O.M.C.C. Five had unilateral O.M.C.C. Four cases were found to have chronic hypertrophied tonsils, and two had atrophied and embedded tonsils. 10 cases disclosed maxillary sinusitis and one patient had bilateral ethmoiditis. One case had a frontal and maxillary sinusitis and one patient had a pansinusitis. Three patients had high blood pressure which was present at variable periods at subsequent visits. Four patients showed generalized ateriosclerosis. One had a marked anemia and the other case had a very high blood sugar.

It is not surprising to learn from the above data that even in a series of only 25 cases that so many etiological factors can be found to exist. It also helps to prove that it is very important to completely and thoroughly analyze each patient in order to come to a definite conclusion as to the causative factor related to the most disconcerting and terrorizing symptom, tinnitus aurium. There is nothing to prevent a patient from having as many as three causes related to the existent complaint. An old and experienced medical scientist once stated concerning diagnosis in disease that most failures in proper diagnosis were due to the want of looking and not because of the want of knowing.

Treatment of Tinnitus Aurium: There are two groups of cases, one without deafness, and the other tinnitus aurium associated with deafness. A very good physical examination should first be made and the general health must be attended to, such as constipation, renal conditions, vascular system, including heart and blood pressure, anemia, functional nervous diseases, neurasthenia, exhausting physical and mental conditions and excesses in tobacco and alcohol curtailed. The diseased and abscessed teeth extracted as soon as diagnosed.

The sinuses and nasopharynx carefully studied and carious teeth and infected tonsils to be attended. Urine should be examined and the various blood tests should be done when indicated surgically.

In cases where there is tinnitus with deafness, pressure on the footplate of the stapes makes for tinnitus so that the presence of any existent pressure should be relieved. In this group are objective signs. In the class where there are objective signs, it is necessary to look into the external auditory meatus where there may be wax, foreign body, polypi, osteoma, etc. Upon examining the tympanic membrane it is necessary to look for middle ear infections, acute inflammation or bulging with or without perforation. In this type tinnitus is pulsating or blowing. When the acute condition subsides, tinnitus disappears. There may be an exudative middle ear catarrh, in which case there is a creaking sound or noises similar to that of running water. Drury[64, 65] states that treatment is unfavorable when the noises do not vary and are continuous. The treatment is less unfavorable when the sounds vary in intensity, and there is temporary cessation; the therapy is favorable when noises are altered by inflation. Tinnitus aurium may be relieved by properly placed incision of the tympanic membrane in interstitial myringitis. E. M. Josephson[66] claims that there are different pathological changes in vascularity. It is necessary to study the various structures associated with the ear drum. A thorough history taken from the patient will give valuable information and this should be done by the examining surgeon himself. In neurosis and neurasthenia, bromides have been used with some relief. Dilute hydrobromic acid, 30 drops three times per day, in a glass of water, has been useful. In patients suffering from high blood pressure, nitroglycerine has been employed. Some have tried counter-irritants placed over the mastoid[67] area. Epinephrin 1/1000 dilution, ten drops in a glass of water one-half hour before meals, have been tested in certain cases. Purging with magnesium sulphate on an empty stomach has been helpful in some instances. Others have tried electro-otopneumomassage with the expectancy of giving relief.

Two types of instruments for mechanical treatment of tinnitus have been devised by Jones and Knudsen[68] for relief of

tinnitus. The pitch and loudness of the tinnitus are first determined by precise measurements. An objective sound is then produced of the same pitch as the tinnitus, but much greater loudness is employed. In this way by bombarding the cochlea with much louder tones there will sometimes be a lessening of the tinnitus. But this fatigue phenomenon does not last. The other type of instrument is a monotone generator. In this type of mechanism an oscillator is adjusted to produce a frequency slightly below that of a tonal tinnitus. In the harmonic generator a broad series of frequencies is obtained. Although this instrument does not cure the tinnitus, yet it may afford relief so that the patient can sleep.

It is Osborn's[69] idea that in cases where tinnitus is caused by overuse of quinine such condition can be prevented by giving three grains of bromide with every gr. I. of quinine. The exception is in pernicious malarial fever attacks where too much of this drug is necessary and an excess of bromide might be administered.

F. P. Strum[71] suggests atrophine sulphate instilled through the perforation of the drum if one is present, or it can be introduced through the Eustachian tube via a catheter using grains V. in alcohol to one ounce of aniline oil. He believes certain paralyzing effect on secretory fibers of different nerves from peripheral sources, such as the tympanum controls the reflex secretion of the endolymph.

Mallison[70] (London) recommends dilute hydrochloric acid, one dram three times per day in some water. On the other hand, H. M. Wherry (London) advises the use of nitrates in cases of high and low blood pressure.

Bruhl,[52] Albrecht and Sabotsky think that the use of radium might be tried. Bruhl's report is optimistic, while Sabotsky recorded one case of tinnitus cured and improvement resulted in ten patients out of twelve. In a series of other 12 cases, however, there was no improvement. Two patients were worse.

McBride[52] is on record for cures of reflex cases of tinnitus by cocainization of the sphenopalatine ganglion. There were two sources of reflex irritation, *i.e.*, one relaxed condition of the temperomandibular joint of one or both sides which occurs when a disturbance exists of a normal closure of the teeth. This joint having been used to a certain force in its bite for

a number of years. With the wearing down or loss of the teeth, principally the grinders, this closure becomes altered with strain on temperomandibular joint which produces somewhat of a subluxation with resultant stretching of the joint capsule. By use of proper plates and checking the joint position by X-ray examinations in cases where this is the cause of tinnitus such symptoms are quickly relieved. The second type and more easily recognized cause of tinnitus can be corrected in case of impacted teeth. When this condition is present its continuation should be interdicted. Many good results have been produced following the extraction of impacted molars.

The writer feels that the future treatment of tinnitus in certain types of unknown etiology lies in properly understanding certain unrecognized toxic agents produced in the intestinal tract which seems to have a predilection for the auditory nerve. Living matter with properties of growth, movement, memory, intelligence, etc., is composed of from 70 to 90 per cent of water. Any change in the amount normally present makes a difference immediately in the activities of the cell. The primary[73] cell contains sodium, potassium, magnesium, calcium, iron, sulphates, chlorides, phosphates, carbonates in all living matter. The amount differs in various cells. Salts are absorbed with water and tolerated, but not in combination in part at least with organic matter. These salts are active in production of vital phenomena. By increasing or diminishing the proportion of sodium, calcium or potassium, skeletal muscles may be made to twitch rhythmically or to remain at rest. Nerve impulses may be made to set up in motor nerves or the irritability of the nerve raised or lowered. It remains to be more clearly demonstrated that the problem of balanced diet,[82] acid and alkali tolerance, vitamin influence has a therapeutic bearing on the factors behind tinnitus and progressive deafness. Cases of tinnitus have shown marked responses as well as migraine and atypical neuralgia which were negative to focal study, have apparently cleared up under nutritional reorganization. Who knows what the new discoveries in the science of physiological chemistry and physiology might unfurl for the application and treatment of otological problems, such as deafness and tinnitus?

M. J. Gottlieb,[70b] in "Deafness Due to Pancreatic Insufficiency," brings to the attention of otologists the following:

"There is no doubt that during toxemia of any origin the sensitiveness of nerves is diminished and when an organ is especially vulnerable greater damage will result. It is possible that we inherit the type of circulation that our ancestors possessed and this may explain the frequency of hereditary deafness. That hearing is suggestive of a relation between auditory acuity and blood vessel integrity. This idea is further encouraged when, on investigating the detailed symptoms of the cases in consideration, we find that the tinnitus is made louder and more annoying, and that the hearing is less acute following coitus. Furthermore, following a short interval after awakening from sleep, the tinnitus is resumed with greater force than at any other time during the day and this lasts for a short time. There is no doubt that profound changes occur in the circulation just prior to and following sexual intercourse and during the transition from sleeping to waking."

Carefully selected cases of tinnitus with nerve stimulus located at peripheral end of the auditory nerve offer a fair prognosis for the cessation of tinnitus after the operation of cutting of the eighth cranial nerve is performed. Wallace[74] and Marriage,[79] after sectioning the auditory nerve, noted that tinnitus was diminished but death followed three weeks after the operation. DaCosta[80] described an operation proposed by Krause and Ballance, who performed it on the right side with success in a case of distressing and painful tinnitus. The auditory nerve was divided. When the cerebellar hemispheres were displaced by sponges the nerves of the posterior fossa were brought into view. The auditory nerve was divided, but no other attempt was made to preserve the nerve of Wrisberg. Five months after the operation the patient was well except for deafness and deviation of the tongue to the left (Ballance[81]). Frazier has divided the auditory nerve for aural vertigo with partial relief.

If after appropriate general and local treatment serious tinnitus still persists, there should be recommended, after proper consultation, in cases where suicidal tendencies are present, the sectioning of the auditory nerve. Proof must be established that the source of the noises are in the peripheral portion of the auditory nerve (Bryant).

The curettement of the cochlea is not worthy of extended consideration in tinnitus, as it is very difficult to totally de-

stroy the cochlear nerve within the cochlea (Green and Lake[76]). Lensmayer, Grunert and Meyerson have described ligation of the carotid, but it has only had a transitory effect on tinnitus. In this procedure there is no destruction of perception of the noises. Its effect is not certain and is not direct. Parry's[78] method has been condemned in his approach to the auditory nerve through the middle fossa. There is some danger of breaking the bone and may result in compression of the cerebellum. The patient was reported to have recovered from the operation and was slightly improved. One year after the operation the patient's tinnitus was considerally diminished, but the hazard is too great in this type of procedure. Where there is organic pathologic changes as fixation of stapes or dense adhesions, the old type operations, such as cutting of the auditory nerve, has not given satisfactory results. But the author is of the opinion that more fruitful work can be done along these channels with proper understanding of cerebral surgery which has recently been developed.

Babbitt[82] describes the separation of the intratympanic musculature and freeing of the intratympanic space had proved the noises in the ear had not become a memory tinnitus in the higher acoustic centers, and he thought some of the slightest handling might be required to keep this more elastic; he also feels that the syndrome of tinnitus aurium is too complex to require any conventional routine treatment. According to his description, the tympanic cavity offers a future surgical means for obliteration of tinnitus and it is futile to assume a memory tinnitus is present unless there is demonstrated the presence of labyrinthine involvement or that the auditory nerve is affected.

Kopetzky[83] recently has described some cases of postoperative mastoid tinnitus. In his form of treatment he advises the use of careful catheterization as many as three times per week, which may be followed persistently and carefully and discontinued when symptoms subside. The gradual return of normal hearing soon is experienced and there is relief of the tinnitus. This is created by bringing back normal amount of air pressure on either side of the drum membrane.

The effect of diathermy upon tinnitus is as incalculable as its use upon deafness. In its milder forms, tinnitus with a

short duration, whether due to simple Eustachian catarrh or to otitis media, soon disappear. But in the continuous, intractable tinnitus of established kind, the results are much less satisfactory, besides being very variable. Immediately following first exposure to the current the noise frequently becomes louder, but it usually subsides in an hour or more, to be followed by a subsidence that lasts sometimes 24 hours (McKenzie). Some authorities are inclined to believe that if lessening does not take place the patients may feel discouraged and do not continue treatments. Yet a second application should be utilized before all hope is shattered, for it has been observed that after two or three applications complaints of the noises being louder do not exist. The general effect[84] upon severe tinnitus is that much more variation in loudness is experienced. At times it is as obtrusive as ever or even more so. At other periods it becomes more hushed as almost to be inaudible. It is the same with hearing as well as tinnitus. The results of treatment of tinnitus with diathermy is very variable in different individuals. The reason for the variation has not been discovered. The author believes that the experiments along the use of diathermy for protracted periods have not been utilized. More patience must be taken over longer spans and careful questioning by the surgeon himself of those whom he is treating is requisite. The medical profession must be careful about permitting technicians to arrange the records and interviewing the patients. It is encouraging to some who have found a very moderate improvement as has been found in several cases. Diathermy is of most service in early stages of middle ear deafness, and out of eleven[84] cases of less than one year's duration, three were cured, four showed improvement and only four were unaffected by this treatment. It has been found that deafness and tinnitus during and after colds in the head, and in ear conditions, in influenzal conditions and those of acute purulent otitis media, should be carefully studied with application of diathermy.

Guttman and Kulvin[83] claim that some individuals using diathermy have accepted patients' subjective statements of improvement in certain types of deafness and tinnitus. These were not under scientific control. The form of treatment pursued was a thirty minute application of diathermy which was employed three times per week. Heavy coil was used. Elec-

trodes placed over the treated mastoid and the opposite cheek. Contact maintained by a bandage. The patients' subjective sensations used in determining how much of current could be used. It was not frequent to use more than 400 to 500 milliamperes. They treated hearing at weekly intervals under standardized conditions. Electrical constants were consistently watched. Their observations in eleven cases of diagnosed otosclerosis where diathermy was utilized, resulted in nine out of eleven patients claiming they could hear better over the telephone and during conversation felt more at ease. But voice and audiometric tests showed no improvement. The other two patients noticed no change in ability to hear nor was there any decrease in noises. It has been found that diathermia has little or no effect on otosclerosis.

Some otologists found that diathermy may cause resorption of exudative products and fibrosis. Middle ear and Eustachian tube may be affected by diathermy beam by placing the electrodes over the mastoid opposite cheek. Both patients[85] who suffered from tubal occlusion stated that sides treated by diathermy were unchanged while the areas treated by use of inflation and application of tube with ephedrine sulphate by Yankauer applicators become normal, and later the side which did not respond to diathermy rapidly began to show favorable signs of improvement with use of ephedrine and inflation therapy. It is, of course, not sufficient evidence but ought to be tried in a series of at least 100 cases. Diathermy[85] treatment in 32 patients, 24 out of which could hear better after checking up on voice and audiometric tests. Three out of the entire number could actually hear much better, the latter three cases benefited were of eighteen months duration or more. But it is to be remembered that spontaneous improvement could occur. Eight cases did not show any improvement after making determinations with voice and audiometric tests. The writer's conclusion is from the facts gleaned from the survey of Guttman's and Kulvin's review that if only 42 patients were tested, it would be insufficient data to disprove the future of diathermy. The author feels that much is to be gained from persistent efforts in the application of diathermy under careful observation by capable and trained specialists in the otological field. No data is to be accepted from unskilled and especially technicians who might be employed in the otologist's office and know nothing of otology.

Coleman Scal[87] studied a series of 60 cases which did not show any other pathologic changes except tinnitus aurium. He used thyroid extract as routine gland substance, administered ¼ to ½ grain three times per day. Patients were watched for overdosage signs. The results were that some reported improvement on the third day and others at the end of the week. About 70 per cent of the patients responded to thyroid and some of these were free from ear noises in two months after treatment was instituted. Others displayed some improvement and continued to come to the clinic regularly. In 10 per cent of these cases thyroid had no effect. In such cases adrenalin substance total gland gr. II, three times per day was given. This caused much improvement but did not act as quickly as thyroid. Ten to 20 per cent of the cases did not respond to glandular therapy except one male patient, who had some relief after use of orchic substance. Ovarian extract gr. V, three times per day, produced appreciatively good results in some patients.

Josephson[88] found, in a fairly large series of cases, the intensity of the superimposed sound required for hearing above the normal threshold for that frequency, constituted an approximately reliable measure of the intensity of the tinnitus. He was able to measure and correct certain types of tinnitus with his apparatus. He also charted the different pitches of tinnitus. The instruments used were a beat frequency oscillator of continuous range with an appropriate attenuator and an amplifier, besides a vacuum tube voltameter or an indicator of power level. Then the fundamental pitch of the tinnitus determined by stimulation with proper intensity over the full range of frequencies. The threshold of the determined pitch overcoming the marking effect of the tinnitus then arranged; it then was compared with the normal threshold of that frequency. The result was the level of tinnitus. The frequency of tinnitus is generally at pitch or pitches where the auditory curve drops off sharply. When the intensity of the superimposed sound was raised to a sufficiently high level, tinnitus was drowned out; there only remained in the ear a rumble that was synchronous with the pulse. The pitch of tinnitus has been found to be shifted by the same factors and therapy that affects similar shifts in the level and cure of hearing. Vascular changes, barometric, climatic differences, galvanization, auditory fatigue, pressure

on the carotid arteries, results in a transient intensification of tinnitus after pressure is released. Relief from low pitched tinnitus was obtained in a great majority of cases rather consistently, especially in patients suffering from spasm of the auditory accommodation mechanism, in cardiac patients with insufficiency when they were digitalized, in anemic patients when proper medical treatment was instituted. In those patients who suffered from high pitched sounds, relief was obtained but not marked in conditions of arteriosclerosis and maladies of hypertension.

Iglauer[89] quoted four cases where tinnitus of objective and subjective types arising from either spasmodic rhythmic contractions of muscles related to the Eustachian tube or to sounds that are vascular in origin. He believed that where a patient's condition is so severe and intolerable, the sounds disappear on light digital compression of the internal jugular vein as well as after torsion of the neck; in such cases the vein might be exposed under local anesthesia and provisionally occluded. If tinnitus stops, the jugular vein ought to be ligated. This, of course, is his opinion. He feels that subjective and objective tinnitus could be modified by torsion of the neck or by digital compression over the carotid sheath. This might be only temporary.

E. D. D. Davis[90] quotes a case where a married woman, age 33 years, complained of aural vertigo and tinnitus of some months' duration and stated that 13 years before she had a bilateral radical mastoid operation. Dr. Davis, after completing the examination, decided to do a right superior and inferior vestibulotomy with curettement of the vestibule. The operation was easily and quickly carried out, but the patient returned at a later date with no improvement of the vertigo and tinnitus. Four months after Dr. Davis had first performed the operation on this patient, she was again admitted to the hospital, where, this time, a left superior and inferior vestibulotomy with curettement was performed. The patient's condition thereafter was much better with no recurrence of her symptoms.

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SCHWABACH'S TEST: THE AUTHOR'S TEST.[1] A COMPARATIVE STUDY.*

DR. B. M. BECKER, Brooklyn.

1. Schwabach's Test: This is based on the fact that in impairment of the auditory function resulting from a perceptive lesion, hearing by osseous conduction is markedly shortened when compared with bone transmission in conductive deafness or in normal hearing.

The test is performed by placing the handle of a vigorously vibrating tuning fork on the mastoid of the affected side in a patient suffering from a unilateral perceptive affection, and when he ceases to hear the sound, by transferring and setting the stem of the still vibrating fork on the mastoid of the unaffected ear and marking, in terms of seconds, how much longer he hears the fork on the normal side. In a patient suffering from a bilateral nerve lesion or for the sake of greater convenience the still vibrating fork is transferred and placed on either mastoid of the examiner, provided the latter is free from any perceptive deafness and a comparison is made between the hearing time of the examiner and that of the patient.

As in all functional hearing tests, great care must be exercised in the method of application and in the interpretation of the results of the examination lest the observer be led into drawing erroneous conclusions.

Since the test is essentially subjective in character in that the examiner must of necessity rely upon the statements of the patient relative to the duration of his sound sensation and since the differential diagnosis is based on the difference of a few seconds between the hearing time of the physician and that of the patient, it is advisable to consider thoroughly all possible sources of error.

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(A) SOURCES OF ERROR NOT INHERENT IN THE TEST ITSELF.

1. Understatement of the hearing time: We must bear in mind, in performing the test, that in making comparisons between the hearing time of the examiner and the patient that on the one hand we deal with a trained observer who is used to the fork sounds and therefore will follow them to a point where they become totally lost and, on the other hand, we deal with an untrained and perchance unintelligent individual who is unused to the fork sound and therefore may follow it only when it is clear and distinct, but when the sound becomes vague and loses its distinctive character, he may assert that he hears it no longer. This apparent difference in the hearing time between one and the other may spell the difference between a right and a wrong diagnosis.

2. Overstatement of the hearing time: The patient may apparently hear the sound longer than he really does, due to an after image of the sound sensation, as in the case of vision when the sensation of seeing an object may still be present when the direct stimulus thereof is removed from the retina.

3. Vibrations mistaken for sound: Some patients mistake sensations of vibration for sensations of sound; especially is this true when they hear the fork sound very feebly or not at all. These last two (after images and confusion of vibrations with sound sensations) often account for the contradictory results obtained on retesting and, if not borne in mind at the time of the examination, may invalidate the test.

4. Normal variation in the duration of sound perceptions: It must be conceded that in persons with normal hearing, all other things being equal, there may be slight differences in the hearing time as there may be slight variations in the hearing distance under normal conditions. What allowance then shall we make, in terms of seconds, for normal variations? It is the writer's opinion that between two and three seconds for Schwabach's test and between three and five seconds for the author's test can safely be made for normal differences in hearing. Anything above these limits must be regarded as pathologic. Obviously to make a differential diagnosis of deafness based on two or three seconds in the one case and three to five seconds in the other between the hearing time of the examiner and that of the patient especially, when this difference is based on the use of one fork, may likely lead to error.

(B) SOURCES OF ERROR INHERENT IN THE TEST ITSELF.

1. In unilateral affections of the perceptive apparatus or in unequal bilateral involvement of the same, this test is unreliable, unless one can eliminate or exclude the normal or better ear from sharing in the process of hearing the sound simultaneously with the opposite one.

2. The difference in the hearing time between the patient and examiner, when a vibrating fork is applied to their respective mastoids, may be due to anatomical or pathological differences in the structure or composition of the cranial bone of their skulls.

3. Any difference in the hearing time of the two may be due to anatomical or pathological differences in the soft parts covering their respective mastoids.

4. The difference in the duration of the sound sensations of the examiner and the patient may be the result of differences of pressure of the fork handle exerted at the points of contact on the two mastoids.

5. A slight inequality in the duration of sound perception of the observer and the patient may result from placing the fork stem at two different areas (within an inch) on the mastoids of their skulls.

6. Any variation in the length of the hearing time of the two may be the result of partial exhaustion of the auditory nerve due to the prolongation of the sound stimulus, thereby rendering the patient less capable of following the sound to its final extinction.

7. Time is consumed in the transfer and adjustment of the fork from the mastoid of the one to that of the other, or to the opposite mastoids of the same skull, thereby weakening or damping the vibrations of the fork.

8. The vibrations of low pitched forks, when used in this test, may be mistaken, by the patient, for sound sensations and therefor tend to confuse the issue.

While some of the possible sources of error enumerated above are of theoretical significance only and can, by careful attention to method be obviated, others are of great import-

ance and constitute definite drawbacks of the test which may seriously interfere with the results of the examination.

2. The Author's Test.[1] This is an extension or continuation of the ideas evolved and the work done in connection with articles previously published, "Hearing by Bone Conduction"[2] and "The Palm and Fork Test."[3] Briefly stated, it is based on the following propositions: *a.* Hearing by osseous conduction is merely an indirect or modified way of hearing by air transmission. When a vigorously vibrating tuning fork is brought in contact with any part of the skull it produces vibrations in the latter and likewise in the air contained in all its cavities. The air vibrations in the ear canals induced by the resonating skull is carried to the labyrinth through the conductive apparatus and there experienced as sound. *b.* Obstruction of the ear canal with a finger will greatly enhance bone conductivity, but closing the auditory meatus by pressing the palm of the hand against the auricle will not only prolong the sound, but will bring about the maximum degree of bony transmission. This increased osseous propagation resulting from palm obstruction will exceed in duration and intensity not only that produced by finger obstructions, but also the prolonged bone conduction resulting from any form or degree of conductive lesions. Therefore, when a strongly vibrating tuning fork is set on one mastoid and the ear opposite is closed with the palm, as described, lateralization will, under all conditions save one (that of advanced perceptive deafness), be to the side of the obstruction. *c.* When a strongly vibrating tuning fork is set on one of the mastoids of a patient and the examiner places his ear in close apposition to that of the former (on the opposite side where the fork is applied) so as to convert his and the patient's ear canals into closed cavities the examiner will hear the fork sound for the same duration of time and with the same intensity as the patient. Only when the patient suffers from a perceptive lesion will the examiner hear the sound longer and the duration of the examiner's hearing the sound longer will depend upon the degree of involvement of the patient's perceptive apparatus.

The validity of the propositions or statements enumerated above can readily be substantiated by simple trial or experiment.

Method of application of the test: The examiner sits or stands facing the patient and presses his own auricle against

that of the latter, thereby converting his own auditory meatus and that of the patient into closed cavities. If the patient's right ear is to be tested the examiner places his own right ear against that of the former, and if the left ear is to be tested the examiner puts his left ear against it. A strongly vibrating fork is set on the patient's mastoid opposite the obstructed ear and he is instructed to concentrate on the sound and to inform the examiner by word or sign the exact moment he ceases to hear it. When the patient no longer hears the fork sound the examiner notes, in terms of seconds, the difference between his own hearing time and that of the patient.

Interpretation of the results of the test: As stated previously, in all conditions save that of perceptive deafness in the patient, the examiner and the examined will hear the sound for the same length of time. Therefore, it follows that if the examiner hears the sound longer than the patient (making allowance between three to five seconds for normal variations in the hearing time of the two), the latter suffers from a perceptive lesion. The degree of involvement can be judged by the number of seconds the examiner hears the sound longer than the patient.

The conditions under which both hear the sound of the fork are so similar that the test can be reversed; that is, the examiner can place the fork on his own mastoid with the patient listening to the sound through the former's skull without in the least altering the results.

The superiority of the writer's test over the Schwabach consists in that the former possesses all the advantages of the latter minus its shortcomings.

1. One of the major defects of the Schwabach rendering it unreliable is the inability to rule out the good ear, in unilateral perceptive deafness, or the better ear, in unequal perceptive deafness, from hearing the sound simultaneously with the involved or better ear. This is overcome in the writer's test. The vibrations in the ear canal obstructed with the palm or with the examiner's auricle are so intensified as to double or treble the duration of the hearing time. Therefore, if the sound is lateralized to the obstructed ear, irrespective of the condition of the opposite ear, the test loses nothing of its value. If, on the other hand, the sound is not lateralized to

the obstructed side (after due precaution is taken to ascertain the patient's ability or inability to do so), it is in itself evidence that he suffers from a perceptive affection in the tested ear.

2. The difference in the hearing time of the examiner and examined due to differences in the structure or composition of the two skulls does not obtain in this test for the reason that both the examiner and the patient hear the sound through the same skull.

3. Likewise any difference in the hearing of the two due to differences in the soft tissues covering their two mastoids is also eliminated for the same reason.

4. Differences in the hearing time of the two resulting from varying degrees of pressure on their mastoids is obviated in this test because the pressure is the same for both, since it is exerted on the same mastoid, by the same individual.

5. Since the fork handle is kept constantly in one place, there cannot be any inequality of hearing for the two resulting from different placements.

6. Since both hear the sound simultaneously no inequality of hearing can result as a consequence of nerve exhaustion on the part of one.

7. No time is lost in the transfer of the fork from mastoid to the other, therefore this factor plays no role in this test.

8. The vibrations of low pitched forks, when used in this test, cannot be mistaken by the patient for sound sensations because of the increased intensity of the vibrations in the obstructed ear.

9. The prolongation of the sound in the obstructed ear (doubling or trebling it) gives the examiner a better chance for observation and making comparisons.

10. Finally it enables the examiner to hear the same sound, under the same set of conditions, simultaneously with the patient.

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RESPIRATORY PARALYSIS IN A CASE OF CEREBELLAR ABSCESS. AUTOPSY FINDING: HERNIATION OF CEREBELLUM INTO FORAMEN MAGNUM.*

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It is a well recognized fact that a tumor below the tentorium may cause sudden death when the increased intracranial pressure is quickly released by spinal puncture. Such a death is caused by pressure against the respiratory centers in the medulla as it is forced into the foramen magnum. This complication following cerebellar brain abscess, however, is rare. I could find only two such reported cases[1, 2] in the literature of the past fifty years. Such cases are so rare that I feel a recent death with the autopsy finding of herniation or forceful jamming of the cerebellum and medulla firmly into the foramen magnum is sufficient reason to justify reporting the following case:

A white girl, age 23 years, was admitted to the emergency ward of the Massachusetts General Hospital with the history of having had a left simple mastoid operation performed two weeks previously in a neighboring city. Two days after this operation she complained of violent headache, rotating dizziness and stony deafness. A left facial paralysis was also observed at this time. A left radical mastoid operation was performed to alleviate this complication and it was in this condition that she was admitted to the Massachusetts General Hospital. Upon her admittance to the emergency ward she was in a wildly maniacal condition, so much so that paraldehyde was given to quiet her.

There was no history of any previous ear trouble. The following signs were noted: markedly stiff neck without Kernig, slight choking of both discs and a left facial paralysis. The tongue was deviated to the right, the right-sided reflexes were greater than the left and left abdominal reflexes were absent. Left lateral nystagmus and ataxia were present and

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*The operative work was done on the Service in charge of Dr. F. L. Bogan. The drawings contributed by Dr. D. K. Judd of the Massachusetts Eye and Ear Infirmary.

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pastpointing of the left hand noted. Headaches were severe and paroxysmal, occipital in location. Pulse ranged between 80 and 100; respiration between 20 and 25. Lumbar puncture was cautiously done, only 7 cc. being slowly withdrawn. The fluid was colorless, at 200 mm. of pressure, and contained

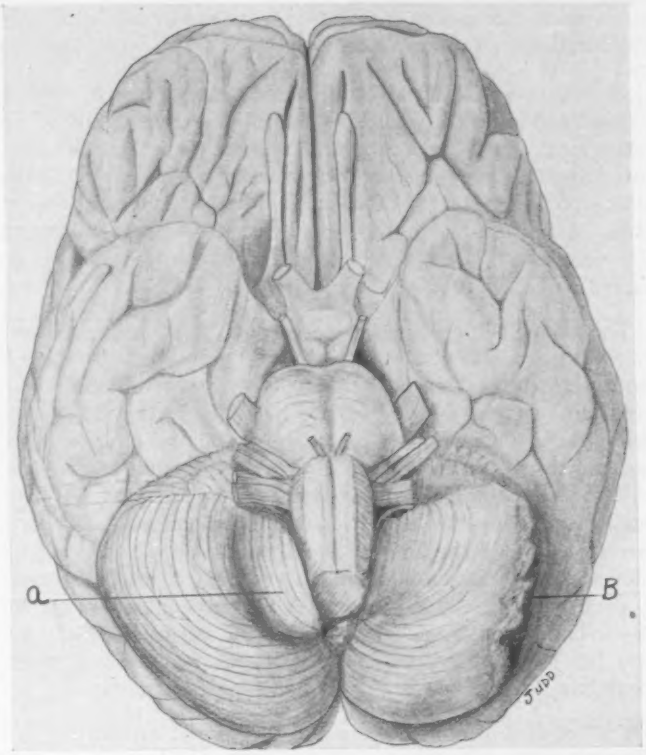

Fig. 1. Drawing of undersurface of brain, showing left cerebellar brain abscess and herniation of right side of cerebellar. (a) Herniation. (b) Abscess.

250 cells, of which 72 per cent were lymphocytes and 28 per cent were polymorphonuclear leukocytes. There was a trace of globulin. Smear and culture of the spinal fluid were negative for organisms. When the effects of the paraldehyde wore

off, the patient again became wildly maniacal, complaining of terrific occipital headache.

The diagnosis lay between meningitis and left cerebellar brain abscess.

After consultation with the chief of service, an exploratory mastoid operation was decided upon in order to settle the question of meningitis, and if this were not present, to explore the cerebellum for an abscess.

A few minutes after the mastoid wound was opened, the patient suddenly stopped breathing and became cyanotic. The heart was regular and forceful. Artificial respiration was instituted and this brought the patient back to normal color. However, upon stopping the artificial respiration, she again became blue. Attention was immediately directed toward uncovering the cerebellum, artificial respiration meanwhile being continued.

The lateral sinus was uncovered and found to be collapsed. It was packed off and an effort was made to go through the sinus wall into the cerebellum in search of an abscess. However, this was prevented by bleeding. The bone over the cerebellar dura, posterior to the sinus, was hard and thick, and because time was an important element, a small area of dura anterior to the sinus was exposed and incised, and a Cushing brain needle inserted. At a depth of 3 cm. in a downward direction toward the base of the skull, an ounce of thin green odorless pus was finally encountered after many attempts. A drain was inserted. Artificial respiration had been kept up during the entire operation. After a period of thirty minutes the patient began to breathe spontaneously. Her color came back and she was put to bed in fair condition.

The organism from the abscess was found on culture to be short chain streptococcus.

An eye consultation now revealed conjugate deviation of both eyes to the right with three diopters of choking in each eye. At times vertical nystagmus was present.

Nine days later the patient suddenly showed five diopters of choking in the right eye and four in the left, associated with projectile vomiting. A second cerebellar exploratory

operation was performed, this time through the sinus wall, but no pus was encountered.

Two weeks later, on account of increasing headaches, a third exploration was performed posterior to the sinus, draining an ounce of thick green pus. At this time diathermy was

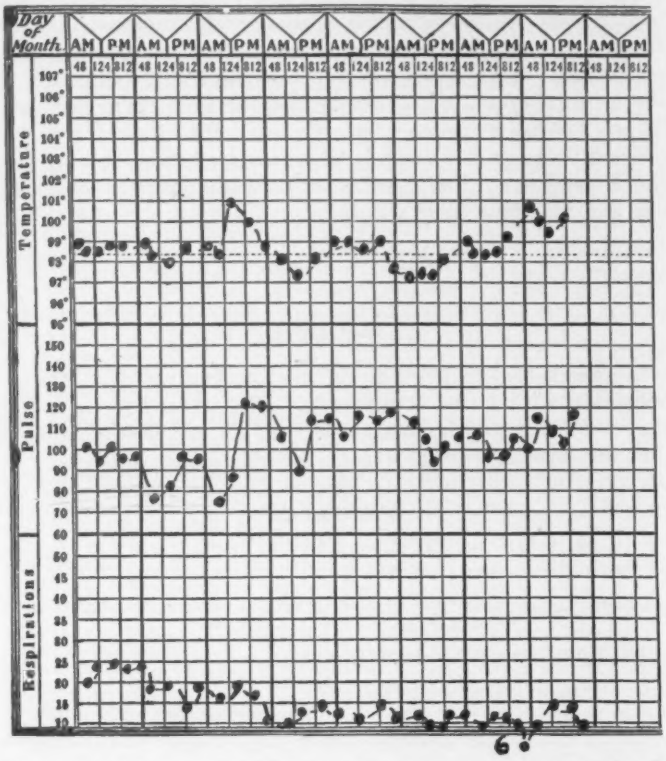

Fig. 2. Note that the respirations have gone down as low as six per minute.

used to remove a cortical cone of cerebellum over the abscess and a Mosher drain was inserted.

For the next four weeks the abscess drained well. However, toward the end of that time the patient's respiration gradually became slower until it reached six per minute, and she died

from paralysis of the respiratory center in the medulla. The heart was strong and regular up to the end. The pulse during the entire illness ranged between 90 and 120. At no time did it go below 70. Before her death a ventricular tap was considered in an effort to release the pressure on the medulla, but her condition would not allow further operating.

At autopsy it was found that the right side of the cerebellum and the medulla had been forcibly jammed into the foramen magnum, producing a herniation or cone of these structures. This accounted for the respiratory difficulty at the original operation, and was also the final cause of death. There was no evidence of meningitis. The large cerebellar abscess was found to have been emptied of pus, showing that the Mosher drain had been in a favorable position for drainage and had done its work well.

Microscopic examination of the cerebellar brain tissue revealed marked edema.

CONCLUSION.

If respiratory difficulties during the course of a mastoid operation with obscure findings is encountered, pressure on the medulla should be considered and an emergency exploration for cerebellar brain abscess should be instituted.

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LINGUAL GOITER.

DR. EDWARD J. WHALEN, Hartford, Conn.

The general subject of abnormalities of development of the thyroid gland must of necessity be preceded by an understanding of the embryology of this gland and those structures associated with it. Including the most primitive, the thyroid gland is present in all vertebrates. Low down in the vertebrate scale the gland takes origin from a median analage growing down from the ventral wall of the pharynx, later being composed of closed follicles containing colloid. Hartley refers to an interesting investigation on certain amphibians of the lower order in which parathyroid bodies arising from the third and fourth branchial pouches are present in addition to the thyroid gland.

In embryos of 1.4 mm., there appears in the middle ventral wall of the pharynx at the side of the fourth branchial pouch a small outpocketing of the thyroid analage. It has become a stalked vesicle in embryos of 2.5 mm., and its stalk, the thyroglossal duct, opens at the aboral border of the tuberculum impar of the tongue. This point is represented permanently by the foramen caecum. Rapidly the duct atrophies and the bilobed gland analage loses its lumen, breaking up into irregular solid anastomosing plates of tissue as it migrates caudad.

The thyroid gland pursues a long and hazardous developmental journey. Appearing in the embryo about the fourth week as the thyroid analage on the summit of the tuberculum impar, it later becomes saccular and continues its course anterior to the trachea and may come to rest: *1.* in the superior mediastinum; *2.* anterior and lateral to the upper rings of the trachea; *3.* in front of the larynx; *4.* somewhere in the structure of the tongue; *5.* it may never leave its point of origin at the foramen caecum.

This classification, which follows that of Lahey, does not include all of the developmental abnormalities of the thyroid

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gland. It is sufficient in connection with the subject of lingual thyroid to refer to the occurrence of accessory and aberrant thyroid masses that may be found at any point in the developmental course from the foramen caecum to the superior mediastinum. Reference only need be made to: *1.* thyroid tissue found along the course of the thyroglossal duct; *2.* masses of tissue found lateral to the internal jugular vein; *3.* intrathoracic thyroid masses which have no connection with the gland proper; *4.* teratoid tumors containing thyroid tissue in the ovary and other sites in no way related to the normal course or position of the thyroid gland.

The parathyroid or epithelial bodies originate as the dorsal diverticula of the third and fourth pharyngeal pouches, each giving rise to a small mass of epithelial cells termed the epithelial body. Beginning as a paired structure, these glands have an analage distinct from that of the thyroid and descend independently of the thyroid. Two pairs of these bodies are thus formed and with the atrophy of the ducts of the pharyngeal pouches, they are set free at the caudal border of the thyroid in line with the thymus analages. On each side in the cranial border of the thyroid are located the pair of epithelial bodies derived from the fourth pouches. From their ultimate relation to the thyroid tissue, they are termed parathyroid glands.

The ultimobranchial body is the derivative of the fifth pharyngeal pouch. With the atrophy of the duct of the fourth pouch, it is set free and migrates caudad with the parathyroid. Occasionally, the hollow vesicle which it forms has been termed the lateral thyroid. According to Lahey, the lateral aberrant thyroid masses arise from these ultimobranchial bodies and are eventually to be found running up each side of the internal jugular vein. Some observers have thought these lateral thyroid masses to be or contain functioning thyroid tissue. They are viewed by Lahey as belonging to the class of teratomas and prone to undergo malign changes.

From these embryological facts we are to conclude that the thyroid gland has a developmental life distinct from that of the parathyroid gland and the ultimobranchial bodies. There is no reason for expecting the same close physical relation between the thyroid and parathyroid glands that exists when the thyroid is in its usual pretracheal position and

which does not exist when the thyroid gland is in aberrant site. As a result of embryological data and clinical observation, it is rational to suppose that a thyroid gland that is found at the site of the thyroid analage is a complete functioning thyroid. There is no reason for thinking that other masses of gland tissue have followed the usual migration to a position lower in the cervical region. After reviewing several cases in which a state of myxoedema followed the removal of a lingual thyroid, Hartley observes that in the majority of such a lingual thyroid is a true ectopia thyroidis and then represents the entire functioning thyroid tissue in the body.

Incidence: Cattell states that the least common of all aberrant locations of the thyroid is the lingual site and relates that the Lahey Clinic discovered two cases in 8,500 operations on the thyroid gland.

H. E. Smyth, in 1911, described three cases that he had observed. In a complete survey of the literature up to that date, he gives a summary of 61 cases. Ulrich, in 1932, in describing a case under his care, was able to report 100 cases from the literature. The great majority of cases have been in females from ages of 15 to 45, the period of functional activity of the thyroid gland. A few cases have been reported in males.

Hickman describes an interesting case. He was called hurriedly to a new-born infant who appeared to be asphyxiated. Palpation of the pharynx showed a mass filling the oral cavity. Death occurred before a tracheotomy could be done. A postmortem examination revealed a lingual goiter completely filling the pharynx.

In Schilling's case, during a routine postmortem examination of a female, age 77 years, there was found a tumor mass at the base of the tongue which on histological examination showed normal thyroid tissue.

Diagnosis: A lingual thyroid is often first seen by the laryngologist who discovers it during a routine examination. More frequently it is brought to his attention by the complaint of the patient, who reports a persistent cough, lump in the throat or varying degrees of dysphagia and dyspnea. Inspection of the pharynx by withdrawing the tongue reveals a smooth rounded mass at the site of the foramen caecum. By

the aid of a laryngeal mirror the gland can be seen in its entirety. Certain observers have described the surface to be nodular, which would be in keeping with the usual changes to be found in a nodular type of goiter. The presence of prominent veins, close to the surface, has been noted by all observers.

The usual size of a lingual thyroid is two cm. by two cm. Its dome-like elevation is from two cm. to three cm. above the base of the tongue. Histological study of tissue removed from the tumor will show normal thyroid tissue and sometimes changes of a colloid or parenchymatous nature. Other conditions of a similar appearance found in this region are carcinoma, sarcoma and gumma. These malignant tumors all admit of histological examination and ready diagnosis. Ashhurst reports the only case of carcinoma of the lingual thyroid that can be found in the literature.

Treatment: To the surgeon who has before him the gloomy picture of a patient who may be condemned to a lifetime of thyroid therapy following the removal of his sole allotment of thyroid tissue, the treatment of a lingual thyroid presents a difficult problem. Smyth, in his splendid review in 1911, estimated that of the cases undergoing operation, 22 per cent developed myxoedema. Recent surveys would indicate that more than half of the cases show signs of thyroprivia within two years after operation.

Not all lingual thyroids require treatment. If we are to believe that the lingual thyroid represents the only functioning thyroid tissue possessed by the patient, then we must expect the usual physiological changes to take place in the gland; namely, enlargement at puberty, variations in size during the catamenia, increase in size and activity during pregnancy.

Rubeli reports a case in which a lingual thyroid increased to such proportions during pregnancy that preparation was made for a tracheotomy. After delivery the gland decreased in size and further treatment was not required. As a result of physiological or pathological activity in a lingual thyroid, signs of dysphagia or dyspnea may require relief. It is suggested by Ulrich that if the patient is not in extreme distress, certain accepted medical measures which are used to control

the physiological enlargements of the normal gland should be used, such as the use of iodine and thyroid extract, which have a recognized action on the thyroid gland and well might care for a temporary enlargement of a lingual thyroid.

The condition described as myxoedema or cachexia thyroprivia may come on immediately following the removal of the gland or may be delayed for months. The usual signs are drowsiness, impaired memory, asthenia, a rough dry skin, and the loss of hair. Some time later the face becomes wrinkled with baggy swelling about the eyes and over the clavicles and suprascapular region. The basal metabolic rate shows an early fall to —30 or lower. In the case reported by Miller, the metabolic rate before operation was 0.0. Seven days after the removal of the lingual thyroid the rate was —30, and the patient went on to a state of thyroprivia. Ziegelman describes a case of lingual goiter that was removed by surgical enucleation. Seven months later the patient returned, complaining of coldness of the body. Her metabolic rate was found to be —28. This patient in addition was found to have an increase in body weight and the jelly-like swellings over the clavicles and scapular.

Up to the present time, the accepted treatment for lingual thyroid has been removal by surgical methods. A study of the literature on lingual thyroid for the last 35 years is a running commentary on the progress of surgery and its modern development. Warren, in 1892, removed a lingual thyroid with a cold wire snare and reported but little hemorrhage following this procedure. Rietlus, in 1904, described a case in a girl of seventeen. The gland was removed through a subhyoid incision and his patient developed myxoedema in a few months.

Meixner, in 1905, found it necessary in a girl, age 22 years, to divide the glossopalatine arch in order to remove the gland. His patient developed myxoedema four months following operation. Allen, Ingersoll and Ludlow, in 1905, reported a lingual thyroid in a girl, age 18 years. Removal was accomplished by the oral route, but a severe hemorrhage occurred, requiring a tracheotomy and ligation of the lingual artery.

Stuart Low, in 1909, advised removal by the oral route after splitting the tongue from tip to base to allow a better

approach. Hayes, in 1912, attempted removal of a gland in a female, age 18 years, by the oral route. Dyspnea and hemorrhage were so alarming that a transhyodian incision was made and the gland removed. His patient developed signs of myxoedema in 15 days, which were relieved by the use of thyroid extract.

The method in general use today is to approach the tumor by the oral route. By means of stay sutures at the tip and on both sides of the base of the tongue, traction on these sutures brings the tongue forward and the tumor into a position where removal by enucleation is readily accomplished. This method was used by Miller in his case reported in 1933. Ziegelman, while stating that surgical removal by way of the mouth is the method of choice, makes the observation that the possibility of treatment by surgical diathermy should be considered.

It is the belief of Miller that the ideal operation would be to allow a portion of the lingual mass to remain after enucleation, hoping that the resulting hyperplasia would be sufficient to supply the thyroid needs of the patient. In one instance he attempted this but was unsuccessful. A short time after enucleation of the gland, a state of thyroprivia appeared in his patient.

According to Ulrich, the use of the electrosurgical knife would facilitate the ease of operation by the rapid control of bleeding. This use of the high frequency electric current is now an accepted surgical procedure, both for the cutting of tissue and the coagulation of bleeding surfaces and individual vessels.

Lee M. Hurd describes the coagulating current used in the treatment of this case as a biterminal damped current of less frequency than a cutting current, which destroys tissue by coagulation. The speed of its action and the depth of the coagulation depend upon the strength of the current applied, and the time it is allowed to act, the nature of the tissue being coagulated and the size of the electrodes used. The high frequency current passing through the tissues creates heat by the resistance of the tissues to the passage of the current itself.

Report of a Case of Lingual Goitre: Miss L. H., age 24 years, single, seen in office July 11, 1933.

Chief Complaint: Difficulty in swallowing and breathing for five days.

Present Illness: Three months ago, patient felt that she had a cold, the only symptoms of this being a constant nonproductive cough. Five days before she was seen, there was a difficulty in swallowing food. Two days later, there was slight difficulty in breathing and phonation was indistinct. There had been a sensation of a lump in her throat for one week. She was seen by her physician, Dr. Edwin C. Higgins, Manchester, Connecticut, who referred her to this office for treatment.

Family History: Father dead, age 56 years; high blood pressure. Mother alive and well. One brother alive and well.

Past History: Nasal and pharyngeal diphtheria at age of four years. No other illness. No operations.

Physical Examination: Temperature 99°, pulse 80, blood pressure 115/80. Weight 93 pounds, which has been her constant weight for past four years. Ears, nose and eyes normal. No exophthalmos.

Throat: At the base of the tongue there was a tumor mass about four cm. in diameter with a smooth surface showing two prominent veins extending from before backward throughout the entire mass. Palpation of the tumor gives the additional information that it is not tender and does not fluctuate. It is well outlined from the substance of the tongue. The tumor practically fills the pharynx. Palpation of the neck is satisfactory due to lack of subcutaneous fat. The trachea can be palpated from the cricoid to the sternal notch and no overlying structures can be felt. As far as can be determined from palpation, there is no thyroid gland in the normal position. No adenopathy present.

Examination of the pharynx and larynx with a laryngeal mirror shows the tumor extending to the base of the epiglottis and filling both valleculae. A section removed for histological study was examined by Dr. Louis P. Hastings, whose report on the tissue was as follows: "Section shows the margins covered with squamous mucosa beneath which the tissue is composed of a fine connective tissue stroma surrounding

islands of typical thyroid acini, many of which are fetal in type and imperfectly developed."

This patient required relief from the dysphagia and dyspnea. Having in mind the unhappy results following the enucleation of the lingual thyroid by other surgeons, it was thought that enough of the tumor mass could be removed by electrocoagulation to relieve her symptoms and, at the same time, allow enough tissue to remain to supply her thyroid needs.

Under cocaine anesthesia, using a bipolar electrocoagulating current, seven punctures were made in widening circles starting at the centre of the mass. These punctures extended to a point four cm. from the periphery of the tumor. The procedure was painless and the patient was allowed to return home. On examination, 48 hours later, the mass had been reduced by one-third. There was some discomfort, especially a constant pain in both ears. There was a superficial slough covering the entire area of the tumor. Five days after the coagulation of the tumor there was a sharp hemorrhage from the mouth which lasted only a few minutes, about 30 cc. of blood being lost. She was seen a few hours after this bleeding and found to have an extensive slough mixed with blood clot covering the area that had been treated. The clot and part of the slough were removed and a small bleeding spot uncovered. This bleeding area was sparked with an electrode, using the monopolar current. Bleeding stopped at once and did not appear again. Fifteen days after the original treatment, the slough had disappeared and there was a granulating surface over the entire area. The tumor was reduced two-thirds from its original size.

The basal metabolic rate was estimated at this time and found to be —3. As there were several small islands of tissue raised above the surface, four more punctures with the electrocoagulating electrode were done on the sixteenth day following the first treatment.

Twenty-four days after the first treatment, the base of the tongue was smooth and slightly raised, showing the outline of the original tumor mass. The part of the tumor extending to the base of the epiglottis had not been treated and remained, filling both valleculae.

Examination on Sept. 17, 1933, about two months after the removal of the lingual thyroid, was as follows: Basal metabolic —3., pulse 80, temperature 98° No tumor could be seen on extending the tongue. By mirror examination, the area about the foramen caecum was smooth and slightly raised. The masses of gland filling both valleculae were present. The patient was entirely relieved of her dysphagia and dyspnea. Her voice was clear and there was no sensation of a lump in her throat. Her weight remains about 93 pounds. No symptoms or signs of thyroprivia had appeared six months after destruction of the tumor.

SUMMARY.

1. A case of lingual goiter is described.

2. It is probable that in patients presenting a lingual goiter that this is the sole thyroid tissue possessed by the patient.

3. The treatment of lingual goiter should be conservative, and surgical enucleation be reserved for those cases presenting extreme dysphagia and dyspnea.

4. More than half of the cases of lingual goiter that have had the gland removed have developed myxoedema.

5. Surgical diathermy with an electrocoagulating current offers a satisfactory method for removing enough of the gland to relieve the mechanical obstruction. By this method a portion of the gland remains and is sufficient to supply the thyroid needs of the patient.

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750 Main Street.

THE INTERRELATIONSHIP OF SINUS DISEASE AND BRONCHIECTASIS WITH SPECIAL REFERENCE TO PROGNOSIS.*

DR. LOUIS H. CLERF, Philadelphia.

Considerable interest has been manifested in this subject with regard to the interrelationship of bronchiectasis and nasal sinus disease. Assuming that there is a connection between these it would be of interest to know if this knowledge has materially contributed in the treatment of these cases from the standpoint of the bronchoscopist and rhinologist, and whether the prognosis has been affected. It is with this thought in mind that I wish to discuss the subject.

The frequent concomitance of bilateral bronchiectasis and nasal sinus infection and the relative infrequency of sinus infection in patients with unilateral bronchiectasis have been observed by many. The original work by Mullen and Ryder, and additional observations by others are substantially in accord on this. While four possible routes of infections are recognized, the lymphogenous and aspiratory routes are, I believe, the most commonly accepted and important. One can very readily appreciate the ease of aspiration of minute droplets of material from the nose and nasopharynx into the larynx and trachea by the simple experiment of instilling a solution of argyrol into the nose. If one then sniffs, as is commonly done by patients with nasal infection, small points of discoloration will be observed on the vocal cords and ventricular bands; later one will find that the tracheal secretion contains argyrol. There are some who believe that sinus infection may be secondary to a bronchiectasis. While this is possible, it certainly does not occur with sufficient frequency to warrant its consideration from an etiological standpoint. The relative infrequency of sinus infection in unilateral bronchiectasis should be definite evidence to support this belief.

Statistics bearing on the relative frequency of the occurrence of bilateral bronchiectasis and nasal sinus infection vary

*From the Bronchoscopic Clinic, Jefferson Hospital, Philadelphia.

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from 55 to 100 per cent. Some observers have found sinus infection in all of their cases of bilateral bronchiectasis. In a series of slightly less than 200 cases of bilateral bronchiectasis I found evidence of sinus disease in 82.4 per cent.

Patients with chronic cough who have an associated sinus infection often are tentatively diagnosed as cases of bronchiectasis on the basis of a Roentgen examination because of heavy hilum markings and bronchial shadows. The bronchoscopic findings in these often are chronic tracheobronchitis with widening of the bronchial spurs. These are unquestionably the result of an enlargement of the peritracheal and peribronchial lymph nodes with a peribronchitis. This peribronchial infection interferes with bronchial movements and cough. Direct aspiration of infection and direct continuity of inflammation accounts for changes in the mucosa, changes in the character and quantity of secretion and interference with ciliary action. These factors in themselves are not always sufficient to cause bronchiectasis; in fact, I question whether they can be considered as more than predisposing factors. The exciting cause, I believe, is acute infection superimposed on the foregoing. Patients, particularly children with nasal infection, are particularly susceptible to acute upper respiratory infections. With these, bronchial infections varying from "chest colds" to pneumonia, are not uncommon. These cases of acute bronchitis, bronchopneumonia or pneumonia are in reality a pneumonitis which runs a brief clinical course and is associated with permanent damage to the bronchial walls. This is the beginning of the bronchiectasis and in the presence of certain bacteria, probably spirochaetes, probably streptococci, may be very extensive.

We should therefore have learned from the frequent relationship between sinus disease and bronchiectasis that a careful nasal examination is necessary in every patient suffering from a long standing unexplained cough with a view of ascertaining if sinus infection is present. Internists also should be aware that chronic unexplained cough can be of sinus origin. The otorhinologist should be always on the lookout for lower respiratory symptoms in a patient who is suffering from sinus disease.

One well may ask, what is the prognosis in the case of bronchiectasis with sinus infection? The prognosis in bi-

lateral bronchiectasis is always unfavorable, irrespective of associated findings. Clearing up of the nasal infection will not greatly influence the course of the bronchiectasis except insofar as it may decrease the frequency of upper respiratory infection and in that way relieve the patient of a certain amount of distress.

The most important consideration in connection with cases of chronic cough of sinus origin is the *prevention of bronchiectasis*. Whereas, chronic bronchitis is considered as the most common etiologic factor in the occurrence of bronchiectasis, it must be recalled that chronic bronchitis is not a primary disease. One must therefore ascertain the cause of the chronic bronchitis. Here again sinus infection plays a very important part and it is in these cases that one can hope to accomplish much if recognition is early and treatment adequate.

Two general impressions exist among a certain number of rhinologists, namely, that the results of treatment in suppuration of nasal accessory sinus disease are of a temporary nature and that they are largely symptomatic for the relief of headache, decreasing nasal discharge, improving drainage and the like. While these are undoubtedly worth while, it is a question whether they are of real value when one is contemplating the management of a case of chronic bronchitis or bronchiectasis with associated sinus disease. I am heartily in accord with Ferris Smith, who stated that the present generally practiced management of chronic sinus disease is unsatisfactory to both patient and consultant, and that it is vitally important that the rhinologist follow a management which will afford proper relief in cases presenting general symptoms of chronic sepsis, in those with local signs and symptoms and in the cases of secondary bronchial infections and asthma. In the cases of chronic bronchitis secondary to sinus infection it is imperative that the cause be removed. We have all seen cases of chronic cough diagnosed as tuberculosis that have completely recovered after appropriate sinus treatment. The internist should always be alert to this from the standpoint of determining the cause of cough and its relief. The rhinologist, treating cases of sinus disease, should be aware that his patient may have a pulmonary lesion which is dependent on the sinus infection and that a failure to secure relief of

the cough is due to inadequate treatment of the sinus infection.

The old adage, "prevention is better than cure," can very appropriately be applied to bronchiectasis. Curing bronchiectasis is a problem for the surgeon and involves radical measures with a high mortality rate. In bilateral cases, a cure can hardly be hoped for. In adults having a definite bilateral lower lobe bronchiectasis associated with sinus infection, one can hope for moderate amelioration of symptoms after proper sinus treatment. If this is done in conjunction with bronchoscopy, postural drainage, intralaryngeal instillation, vaccine therapy and general medical measures, the patient may secure considerable comfort but cannot hope to be entirely free from symptoms.

In children, however, I believe that more can be accomplished by this plan of treatment. My best results have been secured in children. It requires endless time, patience, attention to the general hygienic and dietetic care, and careful and frequent supervision by the rhinologist who should not be allowed to be entirely satisfied with his results. The bronchial dilatations do not disappear. If they are bilateral, surgery cannot be recommended. Active treatment of the upper respiratory infection, improvement of lower respiratory drainage, minimizing the frequency of acute infections and maintaining the patient's resistance at the highest possible level will be productive of results. In this way patients will benefit from our knowledge of the interrelationship between bronchiectasis and suppuration of the nasal accessory sinuses.

1530 Locust Street.

CARCINOMA OF THE MIDDLE TURBINATE. CASE REPORT.*

DR. EDWARD S. LODGE, Los Angeles.

History: In reviewing the literature on this subject for the past 20 years, I find that cases of this nature are not at all common, comparatively few cases are reported. Husik, in the *Annals of Otology, Rhinology and Laryngology,* June, 1922, reports a case of carcinoma of the middle turbinate and ethmoids, two years after radical operation without recurrence. In this article he quotes L. M. Hurd (*Annals of O., R. and L.,* June, 1918), who says that carcinoma of the nose is rare enough to warrant reporting each case.

L. D. Alexander, reporting a case of adenocarcinoma with chronological report in the *Annals of O., R. and L.,* March, 1914, says that adenocarcinoma is essentially a disease of cancerous age, but an early age is possible, as in the two cases he reported, one of 22 years and one of 33 years. His case was radically operated upon and no recurrence after six months.

According to Jonathan Wright, adenocarcinoma is the most frequent malignancy of the nose.

L. M. Hurd, reporting four cases of adenocarcinoma, says Case 1 recurred two years and Case 3 recurred one year after a most extensive radical operation.

William H. Dudley reports a case of adenocarcinoma with exophthalmos, which lived two years and five months after a radical operation, including the removal of the eye.

Louis Klemptner reports a case in a female, age 33 years, of probable origin in antrum and extending to ethmoids and middle turbinate, with no recurrence six months after Denker and ethmoid removal.

L. C. Minor reports a case in a female, age 28 years, origin of middle turbinate, after repeated removal and curettage with no recurrence after one year. Another case in a female, age 22 years, with no recurrence after exenteration of middle turbinate and ethmoids.

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*Read before the Los Angeles Society of Ophthalmology and Otolaryngology, March 26, 1934, Los Angeles.

John McCoy reports a case of origin in ethmoids, with an extension to sphenoid and frontal with no recurrence one and one-half years after curettage and modified Killian.

Etiology: The etiology of carcinoma of the middle turbinate would involve the etiology of carcinoma in any portion of the body. This is still a much debated subject, and will not be entered into in a discussion here. Localized irritation and repeated trauma are the two most prevalent etiologic factors which, in the majority of instances, are found to be the most constant. It is because these factors are found more often in adult life, and much more rarely in youth, that the rarity of carcinoma in youth can be chiefly attributed.

Diagnosis: The important diagnostic phenomena are: gradually increasing unilateral nasal stenosis; mucopurulent discharge; persistent pain; appearance of an indurated ulcer; epistaxis; odor (due to necrosis of soft bony tissue); external deformity and impairment of vision as a result of extension of the disease into the ethmoid cells and orbit; cachexia; microscopic examination of a section removed from the growth.

Prognosis: The prognosis is unfavorable and the recoveries are rare. In the opinion of the authorities on this subject, the outlook is most hopeless. Thus, Carter states: "Prognosis is bad. Only recently, Bloodgood has stated that not one of the 18 cases with malignant disease of the nose operated upon at Johns Hopkins Hospital recovered. There is no authentic case on record where recurrence has not taken place before patient has left the hospital." Modern methods of treatment, however, have brought about a change for the best; and although today the outlook cannot be considered good, it is at least encouraging and bids fair to improve with better understanding of methods and technique. The results of New in the treatment by cautery of that most malignant tumor, the squamous-cell carcinoma; the results of irradiation by Roentgen ray of malignant tumors of connective tissue origin, reported by Portmann and La Chapelle with both sarcomas and carcinoma treated by thorough operative removal of the growth followed by immediate radiation through a wide open wound, all tend to make the prognosis hopeful, even in some of the most neglected cases. In the individual case an opinion must be based on a number of considerations. Accord-

ing to Jackson and Coates, in general, tumors that have been metastasized are inoperable and admit only palliative treatment by radiation, which may retard the growth or diminish pain. Operation may be done on some of these cases, however, when the metastases are of limited extent, and of short duration in the glands of the neck; the cervical lesions being dealt with later either by complete dissection or radiation. It may also be done to relieve pain and distress even when hope of permanent cure has been abandoned. The sarcomas are less malignant than the carcinomas; and of the sarcomas, the fibrosarcoma and the encapsulated round- or spindle-cell tumors are usually only malignant. A good prognosis may be given. In the lymphosarcoma and some round-cell sarcoma, the prognosis should be guarded, though good results may be obtained in many cases. The malignancy of carcinoma varies within wide limits and a prognosis should be based on certain considerations.

Treatment: The treatment must be surgical. According to Husik, who states as soon as a diagnosis of carcinoma is made by the microscope (clinically it is impossible, because in the beginning it nearly always has polyps or polypoid degeneration of the middle turbinate), the best chance for the patient is complete removal, preferably by the external route, and followed up by X-ray and radium. Coates mentions several methods of treatment that are available. The summary of it all, however, is that the treatment of malignant diseases of the nose demands close co-operation of the surgeon with the radiologist and the pathologist.

Copy of letter from attending surgeon (Dec. 11, 1933).

Dear Sir: This is to introduce (name of patient) to you. He was sent to me in February this year complaining of a stuffy right nose, of three months' duration, loss of weight and energy, and a cachectic appearance.

On the 28th February, I removed some of the growth for section and cleaned out his antrum (Caldwell-Luc), which was full of inspissated pus. The enclosed pathological report explains itself.

On the 13th March, I did an extensive external ethmoidectomy and removed the whole of the internal orbital plate as far back as the pterygoid fossa. For ten days he had a large radium cast in the cavity.

On the 28th March, I repaired the wound. There is still a sinus, which I hope to close at a future date. His whole appearance has changed, he has put on weight, his appetite is normal and his mental condition is perfect. At first he had a slight diplopia, but this did not last long. He shoots better than ever. He occasionally has pain over the distribution of the superior orbital nerve, which I suppose, has been caught up in the scar. So far there has been no signs of a recurrence. His wife washes out the cavity and keeps it free of crusts.

They are aware of the condition. I should be most grateful if you would keep an eye on him.

I am yours sincerely,

Mr. Eric Steeler.

33 Harley Street, London, W.1.

Copy of pathological report (March 6, 1933).

Patient: (Name).
Doctor: Mr. E. Steeler.
REPORT ON EXAMINATION OF MATERIAL FROM ANTRUM.
The material sent consisted of (a) a piece of tissue from the nose and (b) ?inspissated pus from the antrum.
Sections of (a) the piece of tissue shows it to be the lining membrane of the nose. In several places there is marked overgrowth of the lining epithelium with downgrowth in the connective tissue.
(b) Here the sections show a structureless mass containing numerous pus cells.
Conclusion: Carcinoma of the middle turbinate.
17-3-33 REPORT ON EXAMINATION OF SEVERAL PIECES OF TISSUE REMOVED BY OPERATION.
Sections show structure of a widespread epithelioma with good deal of inflammation.
T. Jackes, Grosvenor Square, W.1., London.

The patient came to Los Angeles the latter part of December, 1933, and left March 23, 1934, for his home in England. During this time I saw him on an average of once a week. At his first visit I elicited the following history and made the following examination:

Family History: Negative. No history of cancer in the family going back two generations on both the maternal and the paternal side.

Patient's History: Had pleurisy when a young man, and tonsils removed.

Physical Examination: The patient, a male, age 57 years, well nourished, well developed, good muscular and bony development, large stature, skin of good texture, no jaundice or edema, head normal in size and shape, and scalp negative. Eyes with glasses, normal vision, pupils equal and react to light and accommodations; no nystagmus, ptosis or extra-ocular palsies, arcus senilis present in both eyes. Ears: Slight impairment of hearing, hears better in right ear than in the left one. Mouth: Wears an upper plate. Neck: No cervical or thyroid enlargement. Chest well formed, expansion good. Abdomen and extremities negative. Radial pulse regular, volume and tension good. Examination of nose reveals a sinus of five-eighths of an inch long on the upper right side of nose externally, from an external ethmoidectomy. The wound and operated parts inside of nose appeared in good condition. Wiped out a few crusts, no foul odor or bleeding present. Parts examined looked clean and healthy. Blood pressure 110-80, urine examination negative, and blood count practically normal. The condition of his nose at the time of his departure,

March 23, when I saw him last, revealed that the crusting from his nose had lessened considerably. Headaches, which he complained of, were not so frequent. His general appearance is good, has gained six or seven pounds since coming to California, and his nose apparently does not show any signs of recurrence.

1709 W. 8th Street.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Sixty-two candidates were examined, of which number 14 were conditioned or failed.

The Board will hold an examination in Chicago, Sept. 8, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology, and in San Antonio, Tex., Nov. 16, during the meeting of the Southern Medical Society. Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for application blanks.

THE OPTICOFACIAL WINKING REFLEX.*

DR. SAMUEL M. WEINGROW, New York.

In a previous paper[1] the writer described some new facial reflexes. The reaction of the orbicularis palpebrarum, which is of the above group, has been designated the winking response. The sudden closure of the eyelids by the contraction of the orbicularis palpebrarum may be brought about by afferent stimuli from a number of channels. It was shown that stimulation of the trigeminal nerve by tapping the nasal region as well as stimulating the optic nerve by waving an object in front of the open eye produced the winking response.[2] It is also common experience that a sudden loud sound will produce a closure of the eye. In this paper we will limit ourselves to the opticofacial reflex.

Aside from its existence as a distinct reflex the optic facial winking response seems to be of some use in certain clinical conditions. In infants, deaf mutes and in certain mental states where it is difficult to communicate with the patient, the examiner often finds it impossible to determine the presence or absence of vision. This is particularly true in very young infants suffering from cerebral palsies, nystagmus and other conditions in whom cooperation is so little present that even an eye ground examination is impossible.

Reflex Arc and Technique: In his description of the emergency light reflex, McKendree states that excessive stimulation of the retina by light, produces, among other responses, a closure of the eyelids.[3] The light impulse travels from the optic nerve through the superior colliculus and facial nucleus to the tectobulbar and tectospinal tracts.[4] The opticofacial reflex consists of a closure of the lids upon the sudden bringing of an object into the field of vision. The method of its elicitation is illustrated in Fig. 1. The hand of the examiner if placed above, to the side, or below the eyes of the subject and out of his field of vision is suddenly moved across the eyes, thus causing a sudden closure of both lids. If one eye

*From the Neurological Service and Neurological Clinic of the City Hospital.

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is shielded by a cardboard and the other eye stimulated there results a bilateral closure of the lids. The afferent path of this reflex is the same as that of the emergency light reflex, but the efferent path seems to be confined to the facial nerve.

Findings in Normal Individuals: In studying the above reflexes the writer excluded patients suffering from unilateral or bilateral paralysis of the facial nerve since it has been shown that central and peripheral lesions of the same produce an absence or diminution of the winking reflex.[1] One hundred individuals were studied in the manner described above. In all cases there was a bilateral winking response to unilateral and bilateral stimulation.

Fig. 1. Method of eliciting the opticofacial winking reflex.

Findings in Diseased Individuals: Of the cases presenting diseases or defects in the afferent component of the reflex arc, the writer studied about 25. Over 200 individuals suffering from diseases affecting the efferent component of the reflex arc of this reflex were also observed. These consisted of cases of Bell's palsy, peripheral neuritis due to injury of the facial nerve during mastoidectomy or fracture of the base of the skull, and supranuclear and other lesions that occur with general paresis, tabes, meningovascular lues, encephalitis lethargica, meningoencephalitis, amyotrophic lateral sclerosis, cerebral apoplexy, progressive muscular atrophy and subacute combined sclerosis, etc. A detailed description of the behavior of the trigeminofacial reactions in these dis-

eases has been given.[1] In the course of the presentation of cases no detailed description of the efferent mechanism will be necessary aside from such as may occur in persons in whom both the afferent and efferent components of the arc are involved. Because of the varied type of involvement of the reflex arc of the opticofacial winking reflex the following cases were chosen for presentation:

Case 1: J. D., a male, age 76 years, was admitted to the wards of the City Hospital in 1931, complaining of blindness of both eyes. The history revealed that the patient's right eye was removed 40 years ago following an injury and that total blindness of his left eye was the result of a hemorrhage into that eye where he was kicked by an intoxicated man in 1930. Examination of the right eye showed evidences of enucleation. The left eye disclosed a shrunken eyeball with an irregular scar hiding the semblance of a remaining iris. Tapping the zygomatic and other facial regions resulted in normal facial reflex reactions. When the nasal region was tapped, reactions were noted in the orbicularis palpebrarum muscles of both sides. The winking reaction was also normal when a sudden noise was produced showing that the acousticofacial winking response was as normal as the trigeminofacial reactions elicited by tapping certain regions of the face. The optic winking reaction could however not be elicited since either optic nerve was incapable of being stimulated.

Case 2: J. O., a male, age 48 years, was admitted to the Neurological Service of the City Hospital, complaining of dizziness, weakness, occasional headaches and constipation. His past personal history revealed that his left eye had been injured so badly that it had to be removed.

Neurological examination disclosed nothing aside from some evidences of cerebral arteriosclerosis. Tapping of the zygomatic, levator anguli oris and nasal regions of both sides of the face in the proper manner[2] gave normal winking responses. A sudden loud sound also produced a normal bilateral winking response. When the right eye was stimulated in the manner described there resulted a bilateral response of the orbicularis palpebrarum which was absent when an object was suddenly moved in front of the left eye.

Case 3: S. D., a married blind male, age 66 years, was brought to the office complaining of nausea, constipation and

general weakness. The past personal history disclosed that his blindness had been of eight years' duration. One eye had been operated upon six years before.

General examination revealed nothing of note except some evidences of general arteriosclerosis. Neurological examination disclosed unequal tendon reflexes in the biceps patellar and achilles reflexes. The abdominal and cremasteric reflexes were absent. The right supraspinatus reflex gave no reaction while the left gave a lively response.[5] The pupils were dilated and fixed. There was a bilateral optic atrophy and a disseminated chorioretinitis suggesting a chronic glaucoma.[6] The unequal tendon reflexes, facial weakness, absent abdominals, etc., plus a history of having had antiluetic therapy suggested a diagnosis of luetic basilar meningitis and glaucoma. Tapping the nasal region on the right and left sides gave responses bilaterally with a definite inequality and a marked weakening of the left winking reflex. The acoustic stimulation also showed this inequality. The routine test for the optic winking reaction gave no response whatever on bilateral and unilateral stimulation, thus indicating the presence of total blindness.

Case 4: A. B., a male child, age 3 years, was brought to the Neurological Clinic of the City Hospital with the complaint of blindness, convulsions, inability to sit up or talk. The past personal history disclosed that the infant was a premature of three pounds in weight at birth and one of a pair of twins. Neurological examination revealed evidences of congenital developmental defect plus a bilateral corneal opacity. The trigeminofacial and acousticofacial stimulation gave normal winking responses bilaterally. When the opticofacial winking test was tried there was no winking response on either side.

Case 5: A. M., a woman, age 30 years, received a laceration of the right eye with displacement of the lens and escape of some of the vitreus humor. A short time after the injury the acoustic winking response as well as the trigeminal winking response gave normal reactions bilaterally while the optic winking response was completely absent on the right side and present bilaterally when the left eye was suddenly stimulated. A month after the repair the patient gave normal winking responses bilaterally on stimulation of the right eye.

Case 6: G., a white male, age 51 years, has been under the writer's observation for the past five years, during which time he was treated for arteriosclerosis, diabetes, endocarditis and a bilateral cataract. He also presented a general transposition of the viscera.

Neurological examination disclosed no involvement of the nervous system. Tapping of the facial regions gave normal facial reflex responses, and acoustic stimulation gave a normal bilateral winking response when either ear was stimulated. When the left eye was stimulated there was a slight bilateral winking response which was absent upon stimulation of the right eye where the blindness was complete.

Case 7: A. H., a white male, blind, age 78 years, was examined at the Neurological Wards of the City Hospital. The past personal history disclosed that he was operated upon for strabismus of the right eye in 1917, and that he suffered a stroke in 1931.

Neurological examination disclosed a patient suffering from a left hemiplegia, cerebral arteriosclerosis, bilateral optic atrophy and bilateral cataract. Aside from the above the patient presented a bilateral congenital club foot. The trigeminal and acoustic winking responses were normal, but optic stimulation gave no winking response in either eye.

Case 8: J. L., a white male, age 50 years, was brought to the City Hospital, complaining of inability to walk or stand and loss of urinary control of two years' duration. For the past year he also had visual difficulties. He gave a history of having had a chancre in 1912.

Neurological examination showed a static and nonequilibratory ataxia in the lower extremities, absent abdominals, diminution of vibratory and muscle sense in the lower extremities and Argyll-Robertson pupils. All the tendon reflexes were absent except those of the flexor digitorum longus and flexor hallucis longus.[7] Examination of the fundi disclosed central retinitis and optic atrophy. The blood Wassermann was negative; the spinal Wassermann was four plus, and the colloidal gold reaction read 5554320000. A diagnosis of tabes dorsalis was made.

There was no complete blindness in either eye. Objects appeared as shadows on attempting to see, but the left eye

seemed the better. The orbicularis palpebrarum reactions were normal on tapping the nasal regions and on acoustic stimulation, but the only winking response that could be obtained on optic stimulation was that produced by a strong light suddenly brought into view in front of the eyes.

Case 9: J. L., a white male, age 60 years, was admitted to the Neurological Wards of the City Hospital, complaining of blindness, weakness and inability to walk of five years' duration. He gave a history of having had a chancre nine years before admission.

Neurological examination presented a positive Romberg, nonequilibratory ataxia in both lower extremities, Argyll-Robertson pupils which were irregular and unequal, the left being greater, and bilateral optic atrophy associated with bilateral optic neuritis. Vision was completely gone in both eyes. The tendon reflexes were gone except those on the dorsum of the foot.[8, 9] The reactions of these were unequal, those of the right foot being greater than those of the left. The blood and spinal fluid Wassermann tests were strongly positive. A diagnosis of tabes dorsalis was made.

Tapping both nasal regions resulted in normal winking responses bilaterally which were also present and active on the suddent production of a noise. The winking reactions were completely absent when an object was waved in front of either or both eyes.

Case 10: M. N., a white female, married, age 43 years, was admitted to the Neurological Wards of the City Hospital, complaining of a paralysis of the right side of the body and blindness of the left eye of four years' duration. She gave a history of a stroke, following which the paralysis came on and an inability to talk for two months following the coma.

Neurological examination disclosed a patient with a hemiplegic attitude, motor and coordinative disturbances on the right side, exaggerated tendon reflexes of the same as well as absent abdominals and cremasterics on that side associated with a positive Babinski toe reaction and positive heel tap.[10] The tongue was deviated to the right. Cardiac examination showed an auricular fibrillation. The nonprotein nitrogen of the blood was 25 mgm. per 10 cc. The blood sugar was 85 mgm. per 100 cc. The blood Wassermann was four plus.

A diagnosis of cerebral thrombosis of luetic origin was agreed upon.

Fundus examination disclosed a left optic atrophy. The trigeminal and acoustic winking reactions were normal bilaterally, and while both orbicularis palpebrarum muscles responded upon stimulation of the right visual field, there was no winking response in either eye when the left opticofacial winking reaction was attempted.

Case 11: S. R., a colored woman, age 50 years, was admitted to the Neurological Wards of the City Hospital in a disoriented mental state, showing other mental symptoms and not cooperating while being questioned or examined. The relatives stated that she had been in this condition for the past four years and that she had had two stillbirths in the past.

Neurological examination disclosed a left facial weakness, pupils that were fixed to light, bilateral optic atrophy, absent abdominal reflexes, exaggerated tendon reflexes, bilateral Babinski toe signs, bilaterally positive heel tap signs with toe fanning and toe flexion and a bilateral strabismus. The blood and spinal Wassermann reactions were positive and the colloidal gold read 0001240000. The diagnosis was luetic meningoencephalitis. The meningitic involvement though general was most markedly basilar.

Tapping the right facial regions, including the nasal, gave more marked responses on that side than tapping the left. No matter which nasal region was tapped, the winking response was greater in the right eye than in the left. This inequality was also noted when the acousticofacial winking response was elicited. Stimulation of the visual fields gave no winking response.

The above case is instructive from a number of angles. In the first place, some information regarding the condition of the optic nerve is gained when an uncooperative mentally deteriorated person is examined. In the second place, the inequality of reaction between the right and left orbicularis palpebrarum muscles is brought up. The frequent occurrence of facial and particularly winking reflex inequality in syphilitic involvement of the central nervous system has been previously stressed. One might therefore conclude that a

weakness in the winking reaction on one particular side would make this reflex useless for the detection of optic nerve involvement, particularly in syphilis of the nervous system, where this nerve is also commonly affected. This is however obviated by the fact that the winking response is a bilateral one and the reaction of the right orbicularis palpebrarum muscle will take the place of the left in revealing the condition of the left optic nerve should the left orbicularis palpebrarum react only slightly and vice versa. This may also be the case in not only luetic,[11] but also in other affections of the facial nerve.[1]

Case 12: C., a boy, age 16 years, was brought to the office with a history of head injury two months ago; following the accident the patient suffered from a short lasting semicomatose state followed by headaches, dizziness and nausea. Later visual difficulty set in in his left eye.

Neurological examination disclosed some evidence of pyramidal tract involvement on the right side of his body and an atrophy of the left optic nerve. There was an almost complete anopsia in the left eye and the optic winking response was absent on that side and lively when the right optic nerve was suddenly stimulated.

A number of observations were also made of patients during the comatose and semicomatose states of fractured skull cases. In most instances the optic and acoustic facial winking responses were absent while the trigeminofacial winking responses were normal. This occurred before the onset of papilloedema. A case of tuberculoma of the midbrain associated with tuberculous meningitis following a cold psoas abscess was also studied. This patient suffered an involvement of the facial and trigeminal on the same side as the hemiplegia and eye muscle paralysis on the opposite side. The ophthalmologist reported small tubercles in the retina. There was a papilloedema. The trigeminofacial and acousticofacial winking reflexes were absent on the right side and the opticofacial winking reflex was absent bilaterally.

The above cases and discussions fall in line with previous reports,[1] in which attempts were made to establish interpretations of definite reflex reaction associated with the facial structures in instances of central and peripheral lesions. It was indicated that emotional facial reactions could also be

utilized in differentiating between central and peripheral lesions.[12] The recent work of Seckel,[13] in connection with diphtheria, seems to corroborate our previous reports in which a multitude of pathological lesions of the nervous system was dealt with in connection with facial reflex phenomena.

CONCLUSIONS.

1. A reflex method for testing unilateral or bilateral marked visual impairment or destruction is presented.

2. Cases presenting such impairment with the consequent involvement of the opticofacial reflex are reported.

3. Reference is given to cases in whom the efferent part of the opticofacial reflex arc is involved.

4. Illustrations of the three types of facial reflex reactions, *i.e.*, the opticofacial, acousticofacial and the trigeminofacial are also presented.

5. A case of tabes dorsalis is presented in which the opticofacial reflex gives no response while the emergency light reflex is positive. This indicates that the former is the more sensitive reaction.

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1114 Ward Avenue.

PHILADELPHIA LARYNGOLOGICAL ASSOCIATION.

The following officers were elected at the annual meeting of the Philadelphia Laryngological Society, June 5, 1934:

Dr. Samuel R. Skillern, Jr., President.

Dr. Herman B. Cohen, Vice-President.

Dr. Benjamin H. Shuster, Secretary.

Dr. Robert J. Hunter, Treasurer.

Dr. Henry A. Laessle, Librarian.

Executive Committee: Dr. Arthur J. Wagers, Dr. Douglas MacFarlan and Dr. Louis H. Clerf.

NEW MOUTH GAG FOR CLEFT PALATE OPERATION.

DR. HARRIS D. NEWKIRK, Anaheim, Cal.

In doing cleft palate work, I have found the difficulties greatly increased because of the fact that there was no mouth gag suitable for the operation. The accompanying cut shows an instrument, made for me by V. Mueller & Co., Chicago, which is extremely satisfactory for infants from birth to

two or three years of age. The patient is placed in the Rose position with the head over the end of the table. With the handle up, the tongue blade, in several sizes, holds the tongue well up. The lower part is so arranged that the thick hare-lip, whether repaired or not, is easily fitted into the outside groove and the groove with the small barbs is fitted on to the gums. The great advantage is that the center field is then well exposed so one can dissect clear up to the tip of the anterior part of the cleft without the slightest difficulty.

Johnston Clinic.

THE NEW YORK ACADEMY OF MEDICINE.

Combined Meeting of the

SECTION OF OTOLARYNGOLOGY AND SECTION OF PEDIATRICS.

Meeting of February 21, 1934.

(Continued from a previous issue.)

DISCUSSION.

DR. HERMAN SCHWARZ: Before discussing Dr. Mithoefer's paper on paranasal sinus disease, I would like to say a word regarding the results in tonsillectomy. For years I have been trying to get an idea of just how much benefit has been derived by the removal of the tonsils and adenoids. Therefore, in every history where the child has had its tonsils and adenoids removed, the parent was carefully questioned about the reason for the performance of this operation, and also a careful inquiry was made about the effectiveness of this procedure. Of course, in the cases where I recommended tonsillectomy myself, the reasons and the results were always noted. Thus some eight thousand cases were reviewed by my associate two years ago, and although I have not the time to go into the matter carefully, I want to state just one point, that the age at which the operation is advised has a great deal to do with the result for the following two or three years. Stated simply and without any figures, the younger the child the less likely do brilliant results occur. When tonsillectomy is done nearer puberty it is more likely that the following two or three years show improvement. I believe that increase in the underlying immunity plays a greater role than most of us have been willing to admit.

Now as to Dr. Mithoefer's paper on paranasal sinus disease, I feel that I really ought not to discuss it since I know so little about it, or rather so little is known, that it is very difficult to give a clear and concrete picture of what is meant by paranasal sinus disease in children. I remember some twenty years ago being impressed by Onadi's book on the anatomy of the paranasal sinuses in children. Soon after this I asked a young otolaryngologist regarding the noses of a great many so-called normal children and we were astonished at the time to find so many variations from the normal. Since then there has been so much discussion about sinus disease that the patient in ordinary practice coming in to the office for aid in respiratory infections, instead of saying, "My child has tonsils," now says, "My child has sinus." Of course, in a practical way we may divide sinusitis into two broad classes—the markedly acute and the subacute or chronic cases. In the former with pain, swelling and temperature, the diagnosis is easily made, and perhaps the therapy is more or less definite. The subacute and chronic forms, however, I do not quite understand. I do not understand, for instance, the pathology in the subacute or chronic type. You read a great deal about this in the textbooks on the nose and throat, so far as the adult is concerned. Do you in children, for instance, have a loss of the ciliated epithelium? Do you have much bone changes, and so forth? I cannot find much about this in the nose and throat textbooks concerning the adult. I certainly cannot find it in the books on pediatrics. Now, if sinusitis is so common, if every running nose which continues over a period of time is a sinusitis, if every postnasal drip is a sinusitis—mind you, maybe it is—there is certainly a great paucity of pathological findings so far as infants and children are concerned. The mucous membrane lining of the sphenoid sinus down to the mucous membrane of the intestine and the rectum is about the same. There is very little difference except that in certain portions of it additional cells occur, such as the cilia in the sinuses and specialized

cells in the stomach and intestines. I cannot conceive that in an upper respiratory infection, where the lymphoid tissue of the mucous membrane is involved, that the whole length and breadth of this tract does not participate (as I so frequently say on rounds, the whole works is involved). Of course, as far as the sinuses are concerned, we have the ostia which may close off and produce additional symptoms. Thus, as I have repeated so often, it is very difficult to make the picture a real entity and know what it is about. As far as the etiological factors are concerned, I feel that heredity plays a great part, and underlying immunity is very important. You may remove tonsils and adenoids, you may do nasal packings and use nasal drops with relatively very little result in many instances.

The essayist has gone into the question of therapy. Just how much good thyroid medication does I cannot say, for I have had no experience. That coryza is one of the first symptoms of too much thyroid was new to me and very interesting. Personally, in the chronic cases of what is popularly called paranasal sinus disease in children, I have no definite plan. In fact, I must confess I do not know what to do for them. I do feel very strongly about this, that in the acute cases of upper respiratory infections, unless you have definite marked nasal obstruction, the use of argyrol or any other nose drops certainly does not cure the infection and is often a dangerous method of treatment. The use of argyrol tampons and other such like treatment may do good, but I do not know how to judge it. I do not know when these cases are well. The nose and throat man will say, "It looks much better," but a few weeks later the child has a running nose and an upper respiratory infection again. Of course, the proper climate seems of great importance, but as far as the solution of this problem is concerned, we are very far from knowing just what it is all about, at least in infants and children.

Dr. Charles J. Imperatori: I will confine my remarks mostly to Dr. Mithoefer's paper. Regarding the tonsil, I feel that the last word has not been said. I am of the opinion that probably in a thousand years from now we will not be taking out the tonsil. Insofar as diagnosis is concerned, I agree with what Dr. Mithoefer has said. If we could only dry up these noses!

Rather recently I attended the lecture of a distinguished otolaryngologist who was discussing sinusitis in children. In the course of his remarks in describing how to examine the child, he used the expression that the child should be "mummied," meaning that the child should be swathed in a sheet.

A somnolent colleague sitting on my right suddenly roused himself and whispered to me, "Did he mean to mummy the baby?" I could not help but reply, "Yes! dry it up." He being somewhat of a wag, replied, after settling himself for another snooze, "Tut! Tut!"

I feel that in children, especially those under two years of age, nothing should be done in the way of surgery for a great many reasons, but especially anatomical. The antrum is not much larger than a pea, flattened laterally, and is almost on a level with the floor of the eyeball. In the treatment of antrum disease in children under three or four years of age, it is occasionally easier to enter the sinus through the natural opening.

In these cases of acute sinusitis, local treatment instituted early, frequently helps. However, the vigorous local treatment usually aggravates the condition.

Mild adrenalization, mild suction (Sonderman apparatus), warm vapor and then followed by warm liquid petrolatum usually abates the more acute symptoms. Regardless of what is done in the majority of cases, the condition rarely can be aborted.

Air conditioning will greatly help many of these patients, particularly those who are of the allergic type.

Because of the continuity of the mucosa in the nose and paranasal sinuses, each attack of cold results in corresponding inflammatory changes in the lining of the sinus, particularly the ethmoids and maxillaries.

In the majority of instances, drainage is spontaneous and the patient recovers within ten days or so.

I agree with Dr. Mithoefer in his remarks that he considers chronic sinus disease in children a constitutional disturbance.

I agree with Dr. Schwarz regarding the futility of the so-called nasal pack with silver applications. I have stopped their use in my clinic. Regardless of the silver salt, I rather am inclined to feel that the main reason these patients feel better is that the pack is taken out of their nostrils. Insofar as the various polychrome dyes are concerned, I have yet to see any definite result from their use.

Certainly brilliant results have been obtained by dietary regulation and where necessary, the use of certain alkaline or acid producing foods. The use of dilute nitrohydrochloric acid in fairly large doses in some patients results in almost immediate response. Calcium gluconate sometimes acts very well. The use of various iodine preparations and in those hypothyroid patients small doses of thyroid help to bring about relief.

In these children with acutely inflamed ethmoids and edema of the eyelid, surgery must not be resorted to until a trial of the more conservative methods has been used, such as outlined above. Indication for surgery should be immobility of the eyeball. When surgery is done the anterior cells should be uncapped and drainage established. Prolonged surgery in these cases frequently results in multiple abscesses in the muscles, joints, lungs and brain.

DR. WILLIAM MITHOEFER: I would like to answer a few of Dr. Schwarz' questions. It is my feeling that every inflammation of the nasal membranes is accompanied by an inflammation of the lining membrane of the sinuses. Why should the inflammation travel along one pathway only and not creep into the side streets? This statement, of course, applies to the severe inflammations of the nose. There are mild infections which are not accompanied by sinus involvement. The thing which I wish to emphasize is that the inflammation does not commence in the sinus itself, but in the nose or nasopharynx.

I cannot discuss the pathology found in the sinuses of children in any detail. because we do very little radical surgery in children. The pathology is probably similar to that found in adults, except that an earlier stage is represented. The seed is just being sown and this often represents the beginning of a situation recognizable as an operative case when the patient reaches the age of 18 or 20 years. We feel that this conception could be substantiated if we operated on many of these children and had the opportunity thereby to examine the pathological specimens.

I agree with Dr. Schwarz that nasopharyngitis is a generalized infection. Usually the young patient has still other conditions present. If one could look into the colon, the same kind of inflammation would probably be seen and likewise there is probably a congestion of the appendix present also. It seems to me that this is the explanation for the fact that the abdominal surgeons are busier in the winter and spring with appendectomies than at any other time. The necessity for operation is the result of the infection.

Answering another of Dr. Schwarz' questions, I would say that coryza occurs because of the iodine content in the thyroid extract which produces a form of vasomotor coryza. Non-specific protein therapy in the form of omnadin is preeminently indicated in acute infections. We do not make it a rule to use this preparation as a therapeutic adjunct in chronic cases because its constitutional effect is that of a testing dose to stimulate the patient's own power of combating the infection. This comparatively slight increase in resistance is all that is needed in most acute conditions to turn the tide in favor of the patient, whereas, in the chronic cases this same increase makes very little difference.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of March 21, 1934.

Prickle Cell Epitheliomata of the Ear With Involvement of the Facial Nerve in the Tympanum. Dr. Ward C. Denison.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. THOMAS J. HARRIS: Mr. Chairman, I think this case deserves a word of discussion. It is excellently reported. The reporter has studied the literature very carefully and developed what I think most of us know, that it is a very rare and unusual condition. I have nothing to say except to congratulate him on the care with which he has done the work. However, I do want to raise a question, namely, the question which is a fairly large one, whether such a case as this warrants operation. I am not at the present moment conversant with the literature so far as cures are concerned, but certainly so far as my own knowledge is concerned I do not know of a case similar to this that has been cured by radical measures, such as the reporter recommends, followed by radium. If such a case presented itself to me with the findings he has given us preoperatively, I think I would consider very seriously several times the wisdom of disturbing the case because I think it is true that the outcome of these cases is death, although he does not know the outcome in this particular case.

DR. DENISON: In answer to Dr. Harris' question, I cannot state whether it was advisable to operate upon this case. The cancerous growth had advanced rather rapidly and the facial paralysis had developed. Of course we did not know the extent of the diseased process. The entire tip region was a sequestrum. The growth had extended, causing a crevice which extended down, involving the facial nerve. As I said before, I am sorry that we were unable to follow the case, and that I do not know whether or not the patient is living at the present time.

We have another case, diagnosed as prickle cell epithelioma, which is being treated in practically the same manner, but at the present time it is too soon to report the results. This particular case has no involvement of the facial nerve.

Otitic Hydrocephalus With Report of a Case. Dr. Clarence H. Smith.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. S. P. GOODHART: The subject this evening is a very important one and I am very glad to have an opportunity to briefly discuss this paper. I saw this patient at the Morrisania Hospital with Dr. Smith. We are all very much concerned, I am sure, before going into a skull and we are very glad and welcome anything that will determine for us whether or not we are dealing with a brain abscess. What Symonds has termed "otitic hydrocephalus" and by some others as "hypertensive meningitis" has very wide implications. It seems to me its recognition is becoming very much more frequent. I think

we have overlooked it heretofore. We have gone in and operated when there was really no indication for operation. Certainly, otitis media gives rise to some intracranial activity that produces a very marked increase of the cerebrospinal fluid. Dr. Smith mentioned that the autopsy findings indicate an ependymitis or chorioiditis. I think that is not always true. Just in a few instances I think that pathology has been determined, and it is not actually certain whether it is an increase in the fluid formation or whether there is a decrease in the absorption. However, the fact is that we get very definite symptoms, and symptoms that would ordinarily cause great concern and make us feel at times that we are undoubtedly dealing with an abscess. For instance, It is not so rare to find in these cases, where the pathology is entirely one as Dr. Smith said, of internal or external hydrocephalus or both, it is not uncommon to find such symptoms as involvement of the VIth nerve on one side, or there may even be aphasia or involvement of the pyramidal system with a hemiparesis. These things, of course, in themselves would bespeak a localized process, namely, an abscess.

One point of differentiation is very definite, namely, examination of the cerebrospinal fluid. The findings in a hydrocephalus, uncomplicated, would be, as Dr. Smith stated, an immediate increase in the amount of cerebrospinal fluid with pressure, a clear fluid, and, above all, normal protein and no cells. On top of that we find the alarming symptom very often of a progressive papilloedema, sometimes with retinal hemorrhages. It seems to me that the only way we can make a differentiation is by examination of the cerebrospinal fluid, and if that fluid is without cells, with normal protein, we can feel fairly safe that our procedure should be no intracranial interference but simply release of the cerebrospinal fluid by lumbar puncture. If there is no relief, doubtless the proper procedure is to do a subtemporal operation and release the fluid by puncture of the ventricles. As Symonds and others have advocated, regardless of which side is involved, the puncture should be done on the right side, in a right-handed person, of course necessarily thinking of the possibility of aphasia if the approach is on the left. Then with a small button trephine one might go in on the other side.

I think the recognition of this condition, otitic hydrocephalus, is of much importance. It sometimes becomes very difficult to make this differentiation, as in the case Dr. Smith has presented, where we were undoubtedly dealing with meningeal symptoms for the first few weeks. Following recovery from the meningeal symptoms the patient had papilloedema, headache and very marked increase in cerebrospinal fluid pressure. There was, however, no increase of protein and the cell count was reduced to normal. We have two types of cases to consider; in one there is no evidence of meningeal involvement and we have the typical signs of intense headache, developing papilloedema, increase in pressure with no changes in the fluid. Nausea and vomiting may occur and are not infrequently seen in typical cases, especially where there is an involvement of the middle ear or the mastoid without any further involvement of the sinus or labyrinth, but purely a reaction of the otitis media.

Dr. Smith's patient demonstrated the difficulty of making a differentiation. There was a question of whether an intracranial exploration should be made or whether the conservative measures indicated in otitic hydrocephalus should be followed. Dr. Smith did not operate; the final result established the correctness of his procedure. The pathology and symptomatology seen in otitic hydrocephalus impresses one as almost identical with what appears as a post-traumatic concussion syndrome.

The Window Operation for Hematoma Auris and Perichondritis With Effusion. Dr. Robert C. Howard.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. RALPH ALMOUR: Mr. Chairman, my experience with Dr. Howard's procedure is limited to four cases, three cases of suppurative perichondritis following mastoid surgery and one case of hematoma following a blow to the ear. I heartily recommend the procedure in all these cases for the cosmetic result you get. In suppurative perichondritis you can make as many windows as you require in order to drain the pus. If your pus is present, both on the external and on the internal surface of the ear, the pieces of cartilage which have been destroyed can be removed through this window, adequate drainage provided and a very nicely shaped auricle obtained as the result.

I cannot say any more except to bring out one point which I believe Dr. Howard did not have time to mention. That is this: It is surprising to see the rapid collapse of the edges of the window within two or three days after the operation. One would believe that the defect made by removal of this tissue would remain as a gaping wound, but because of the swelling which is present at the time the window is made, the window appears much larger than it really is. As soon as the clot or pus is evacuated, the two lips immediately collapse and within three days you have what will be a fine hairline incision.

DR. J. COLEMAN SCAL: Dr. Howard's operation for hematoma auris is simple and unique in its originality and presents a technique that has never been described before.

Hematoma auris has interested me for a long time as a number of these cases have come under my care. There is nothing in the textbooks on this subject except a suggestion for aspiration and pressure, incision and drainage. With this treatment the majority of these cases resulted in disfigurement. If these cases are seen early and Dr. Howard's technique be applied, the results should be uniformly perfect and the normal outline of the auricle should be restored. If the window is wide enough, there is no necessity for packing or tight bandaging. A single sterile gauze pad, which may be held in place by adhesive straps, is sufficient and should not be changed too often.

In performing this operation I was struck with the disappearance of pain within 24 hours and complete healing within ten days to two weeks.

DR. SAMUEL J. KOPETZKY: I believe, as I have said at previous meetings, that when any of us can support a new procedure that is being presented here we should put our experience on record. I am putting on record my experience and my observation of this work by Dr. Howard.

This operation by Dr. Howard is a fine piece of technique. It is simple and it accomplishes the results. I want to bear testimony to that and to the ingenuity with which he devised this procedure. It is better than anything that has heretofore been recommended for this condition.

Avulsion of the Vocal Cord. Dr. John M. Loré.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. DAVID H. JONES: Mr. Chairman, ladies and gentlemen: I went to St. Vincent's Hospital and saw the work Dr. Loré did on the cats, and I think he has brought out the subject very clearly. As he said, do not try it on everything you see, but try it on these polypoid (not polyps) conditions we find in the larynx. The case he mentioned is one of Dr. E. Ross Faulkner's on which a laryngofissure was done in 1926. This case has been seen in the clinic by any number of men, and it was very difficult to say which cord had been removed.

DR. CHARLES J. IMPERATORI: Mr. Chairman, ladies and gentlemen: I have had no experience with this new operation of Dr. Loré, but it is very refreshing to hear something entirely new on the subject of the larynx, particularly this question of stripping the cord.

It would seem to me in analyzing this briefly, that what Dr. Loré did was to strip the cord, and maybe in some instances he removed all of the cord down to the muscles, but the vocal process was not interfered with. I think that is the answer to these good looking larynges that were presented here tonight, that the vocal process was not interfered with. Regarding this question of regeneration of the cord, which we formerly thought did not occur, apparently it does occur. Insofar as the indications for the operation are concerned, it is possible that in these hypertrophies of the vocal cords, this operation will become the operation of choice.

DR. M. C. MYERSON: Mr. Chairman and gentlemen: I am a little confused as to whether Dr. Loré is speaking of one or two types of experimental work. He speaks of removing the cord and he also speaks of stripping the cord. If you remove the cord that entails the mucosa, the fibroelastic layer and sometimes the thyroarytenoid muscle beyond, and you will not get a regenerated cord that will function as well as the original cord. I say that advisedly because when I was at the Mt. Sinai Hospital the laboratory asked us to remove the vocal cords in the dogs so that they would not bark. They did not bark immediately after the removal of the vocal cords. If any of the staff are here I am sure they can corroborate the fact that they did bark with a hoarse and changed voice later. In other words, we removed their cords and they did regenerate, but the regenerated cord six months later was not the same cord nor did it function in the way it did originally or in the way a normal cord would. In other words, do not pick out a good public speaker or singer if you want to be radical in this field.

This work is commendable and I am very strongly for it. My belief is that hyperkeratotic papilloma should be treated as a strictly precancerous lesion and should be treated by thyrotomy. Anything short of thyrotomy is the same as treating a small localized carcinoma of the vocal cords, as has been advocated by some laryngologists in the past, by endoral methods that are not acceptable.

As to the question of the use of ether or local anesthesia, my experience with vocal cord work, particularly if you are using direct instruments, is that it is pretty difficult to fix and operate upon the vocal cords under general anesthesia. If you are going to do the amount of work Dr. Loré speaks of, by all means perfect yourself in the use of the suspension laryngoscope. Dr. Lynch, of New Orleans, was able not only to remove a small vocal cord nodule or small vocal cord polyp or any type of benign growth, but he could take a fine knife and enucleate that growth by splitting the mucous membrane over the growth, going down on the cord and cutting away the redundant mucosa. So one can, if one perfects himself in the use of the suspension laryngoscope, do accurate and very fine work. I do not think any instruments today will give you the accuracy that the laryngoscope will give you, regardless of the type of approach or type of anesthesia. If one looks through the textbooks one will see such a great variety of instruments for use on the vocal cords that one is greatly impressed with the fact that there is no unanimity of opinion as to procedure. When it comes to the use of an elevator after tonsillectomy, there is the Hurd elevator. There is no improvement upon it. But when it comes to vocal cord work, there is no unanimity of opinion and we have as yet to find the ideal instrument.

DR. LEOPOLD GLUSHAK: The question as to what part of the cord Dr. Loré strips or advises to strip is important. Is the epithelial layer or muscle fibre stripped? To get a clear understanding of Dr. Loré's work and suggestion, one must remember the anatomical arrangement of the vocal cord. A vertical section through the cord shows it to consist mostly of thyroarytenoid muscle

covered by a squamous epithelial covering under which is a loose, meshy layer. This loose layer, which normally is only a potential space, becomes edematous and is very susceptible to the provocation of all sorts of pathological conditions. A stripping of the epithelial layer in a pathological condition of the cord is therefore carried out very easily and the regeneration is also very rapid and uneventful. We have had this experience, during several years, in cases of papillomata, cord nodules, cystic swellings, when the swelling along with a long strip of the epithelial covering of the cord was removed at one time. This procedure was carried out under complete cocainization of the larynx through indirect laryngoscopy. In cases where the hoarseness was very marked a clear voice was immediately obtained.

If an injury takes place within the muscle, whether pathological or traumatic, permanent damage is left and evidence thereof is seen in the form of an indentation or fixation of the cord, due to a fibrosis having taken place in the injured muscle.

No analogy between the larynx of the cat and the human can be made since the same thyroarytenoid muscle does not act and perform the same complicated functions. The disposition of the various bundles in the muscle gives different actions, which bring about variations in pitch, finesse of tone, in the human voice in contradistinction to that of the animal. In the latter you merely have two muscle bundles in the two cords that come together and produce a fixed tone, harsh and characteristic of that animal, e.g., in the cat, the high pitched tone. In the monkey, for instance, the vocal edge is very fine and the cords though resembling those in the human, come together very firmly, like the two semilunar heart valves, in order to more firmly shut off the air from the chest, so as to enable it to hold its breath over a long period, which is necessary when it goes through its climbing actions. In these animals, injuring the vocal muscle produces very little hindrance to voice production since they do not require the voice in the different forms as the human. The suggestion of straightening out a bent cord as we see in internus paresis, by punching a piece out of the cord from the upper and lower ends of the bowed cord does not seem to me very feasible. We must remember that the entire musculature of the larynx is really a sphincter, divided up into portions through the insertion along the inner circumference of this sphincter of the arytenoid cartilages and the attachment of some of the fibres to the thyroid cartilage.

This sphincter is split up into various bundles which at certain times and under certain circumstances act independently. Where we have a bowing of the cord, an internus paresis, the internal fibres fail to act and approximate the cords thoroughly. This is usually a dysfunction and should be treated phonetically. In treating this condition, I usually advise the patients very crudely to bear down and grunt as if they were constipated and in this way get the entire thyroarytenoid muscle or sphincter to act. By punching out a piece from the bowed cord above and below in order to straighten it, one cannot expect the action of the muscle to improve. As a matter of fact, a grave and permanent injury would thereby result. In the case of the cat, one can punch out pieces of the muscle fibres and the animal will again be able to meow and the dog, respectively, to bark. In the case of the human, a singing voice would be crippled through such a procedure.

In think Dr. Loré's suggestion of stripping the cord is a splendid one. The covering of the cord regenerates perfectly and the procedure is useful in cases of chronic edema with thickening of the cords, as well as papillomata and other benign, sessile growths. It can be done in the office and the patient may go home after resting a few hours, and in two to three weeks the epithelium of the cord is completely regenerated.

DR. EMANUEL M. JOSEPHSON: I wish to ask Dr. Loré to repeat, for purposes of clarity, his description of the depth of stripping of the vocal cords in his clinical cases. Did I hear correctly that he stated that the vocal ligament and the thyroarytenoid muscle fibres were removed in the process of stripping?

Dr. Loré mentioned stripping the epithelium within the ventricles. It would be interesting to know if this process resulted in an annoying dryness of the cord, due to damage of the ventricular glands.

DR. JOHN M. LORE: The hour is late and I do not wish to enter into any acrimonious discussion. In the first place, as far as the anatomy of the cord is concerned, Negus describes the vocal cord as consisting of elastic tissue which is covered by squamous epithelium, and acting as a covering to the arytenoid muscle. Occasionally you will find a few strands of muscle, viz., the aryvocalis muscle. The vocal cord is really the free edge of the conus elasticus, which comes off from the upper surface of the cricoid. This conus elasticus comes up toward the larynx, is attached anteriorly at the anterior commissure, and posteriorly to the body of the arytenoid and the vocal process. As to the question of the thyroarytenoid muscle, it is not the cord but the basis of the cord. What the doctor said about its being a sphincter is perfectly true. I have a few slides myself showing the cross sections of the cord which I did not have time to show.

In the cases we have done, where the lesion was apparently confined to the mucosa, we have simply stripped the mucosa, but where we had to contend with the hyperkeratotic papilloma and suspected carcinoma, we felt we wanted to go deeper and in these cases removed some muscle with the cord. In the sections, the muscle was included in the pieces removed. I do not want to advocate stripping of the cord as the treatment for carcinoma of the cord. I certainly do not want to do that. But, in the hyperkeratotic papilloma, where it is a moot question whether or not it is cancerous or precancerous, the end-results justify the method employed. Are you going in and do a bilateral thyrotomy for this condition? We are just feeling our way for the time being and until something arises to make us feel differently we feel that we should use the method described. As far as suspension laryngoscopy is concerned, there is no question that it is wonderful, but it is not justifiable in all these cases. Why resort to this method when the use of the anterior commissure speculum gives you the desired exposure?

As far as the question of producing a bowed cord, we did not only remove mucosa but muscle. This is not the last word by any means in this work. This paper is only in the nature of a preliminary report, although the work has extended over a period since 1920. I left out the question of function of the larynx deliberately because that is a study in itself. I wanted to present this question of removal of the cord in diseases of the cord. I do not want to go into anyone's larynx unless he has something wrong with his cords. Then he is no more a singer, he is just a man with a bad voice and a diseased larynx. If you can improve his voice, all well and good. After all, the end-results speak for themselves. You can theorize all you like about it. We had a lawyer and sent him back to the bar, and a school teacher back to teaching school. The fact that she had epithelial tissue removed and that it showed fibrous polyps ought to be the answer.

(To be continued in a subsequent issue.)

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Fifty-Sixth Annual Congress of the American Laryngological Association was held at the Hotel Cleveland, Cleveland, Ohio, on June 7, 8 and 9, 1934, under the presidency of Dr. George M. Coates. The program was carried out as planned; all those scheduled to read papers were present. The number in attendance was 65; 45 Active, 16 Associate, two Corresponding, two Emeritus, and 14 guests.

The following were elected to Active Fellowship:

Dr. John Walter Carmack, Indianapolis, Ind.
Dr. Frederick A. Figi, Rochester, Minn.
Dr. William E. Grove, Milwaukee, Wis.

The following were elected to Emeritus Fellowship:

Dr. John F. Barnhill.
Dr. J. Payson Clark.
Dr. Virginius Dabney.
Dr. W. Scott Renner.
Dr. Robert Levy.
Dr. Harmon Smith.
Dr. Joseph H. Bryan.

The deRoaldes prize of a gold medal was awarded to Dr. Harris P. Mosher, of Boston, Mass. The Casselberry prize was awarded to Dr. Anderson Hilding, of Duluth, Minn., for his thesis, "Studies on the Common Cold and Nasal Physiology."

The following officers were elected:

President, Dr. Dunbar Roy.
First Vice-President, Dr. Gordon B. New.
Second Vice-President, Dr. Samuel McCullagh.
Secretary, Dr. William V. Mullin.
Treasurer, Dr. James A. Babbitt.
Librarian, Dr. Burt R. Shurly.
First Councillor, Dr. Francis Packard.
Second Councillor, Dr. Joseph B. Greene.
Third Councillor, Dr. Gordon Berry.
Fourth Councillor, Dr. George M. Coates.

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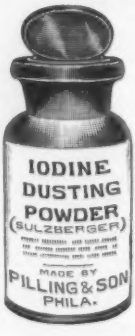

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